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CONGENITAL HYPERTROPHIC PYLORIC STENOSIS IN THE AFRICAN

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Congenital hypertrophic pyloric stenosis in the African appears to be an extremely uncommon condition, since very few cases in the African have been reported in the literature. Ladd, Ware and Pickett[1] state that they have noted no particular racial disposition, and ascribe the fact that they have never observed a case in a Negro infant to pure chance. Donovan[2] reported 2 cases in Negro infants, and later[3] he states that he has found the condition in all races and nationalities and he has seen about 2 Coloured babies with this condition in each 100 cases. Many authors have stated that the condition is rare in Negro infants. Benson and Warden[4] describe the condition in 63 Coloured infants out of 707 consecutive cases reviewed by them during a 16-year period from 1940 to 1955 at the children's hospital of Michigan.

Before 1955 the condition had not been described in the African infant. Shepherd Wilson and Gelfand[5] described the first case in an African male aged 2 months. The following year Luder[6] reported another case in a male Ganda infant aged 1 month. Since then, at Johannesburg, Griffiths[7] has described 1 proved case seen at Baragwanath Hospital in a 1-month-old Bantu male and has stated that a single case, which was treated medically, was seen at Coronation Hospital. Hamilton[8] stated that the literature appeared to contain reports of only 4 cases of congenital pyloric stenosis occurring in Africans, and he described another proved case in a male Dama aged 1 month. Unfortunately the references to these 4 cases were not published with his article, but it would appear that he was referring to the 4 cases described by the abovenamed authors. Thus to date only 5 cases, 4 of which were proved by operation, have been described in Africans.

During a period of 6 years at King Edward VIII Hospital, Durban, this diagnosis has been made on 3 occasions in the Bantu paediatric department. This department has a very large turn-over, both in the in-patient and out-patient section, and the fact that only 3 cases of pyloric stenosis were diagnosed out of an admission rate of approximately 5,000 cases a year, is an indication of its rarity. It seems unlikely that the diagnosis is missed, for the condition has never been discovered as a chance finding at autopsy.

As the condition is usually slowly progressive and does not cause sudden death, presumably the affected infant at some stage would be brought to the out-patient department. The African mother has become increasingly hospital-conscious, and thousands of infants attend for such complaints as failure to thrive, vomiting and constipation; this latter symptom being particularly distressing to the enema-conscious African mother. It is unlikely therefore that cases of pyloric stenosis in any numbers are dying outside the hospital.

There was 1 proved case in each of the years 1953, 1954 and 1956, and these are summarized briefly as follows:

Case 1

A 4-week-old African male was admitted on 24 June 1953. He was a first-born child, his birth was normal and he was breast fed. His birth weight is unknown. The mother, a pure African who came from the kraal, was extremely 'raw' and was unable to give a reliable history even with the help of an interpreter. It appeared that the infant was subject to persistent vomiting and constipation, but the duration could not be ascertained. I was able to interview the father and was satisfied that he too was a pure-bred African.

The infant weighed 6 lb. He was small and miserable, and there was depression of the fontanelle, but skin turgor was satisfactory. All systems were apparently normal. No peristaltic waves were observed after feeding and no mass was palpable. Test feeding was carried out. After 48 hours it was obvious that the infant was vomiting most of what he took in. The vomiting was not projectile in type, but on the following day on 1 occasion it was noticed to be so. Still no mass was palpated. The stomach was washed out and Eumydrin (1:10,000 solution) was given in a a dose of 2·5 c.c. 20 minutes before each feed. For the next 48 hours the vomiting lessened and the infant appeared to be improving. At this stage, after a feed, gastric peristalsis was visible and a pyloric tumour was palpated. After this examination projectile vomiting occurred again. Eumydrin was tried for a further period of 48 hours. Breast milk was now inadequate so that complementary feeds were required. Since dehydration was developing and practically nothing was being passed *per rectum*, operation was deemed advisable. The diagnosis of pyloric stenosis was confirmed at operation and Rammstedt's pyloroplasty was performed.

The post-operative progress of the infant was satisfactory for 16 days when, unfortunately, he developed bronchopneumonia which failed to respond to antibiotic treatment, and he died 2 days later.

Comment. This case tragically illustrates the danger of keeping these babies in hospital longer than is absolutely necessary, because there is the ever-present risk of cross infection. In this case we were influenced by the fact that the

mother was primitive and had to return to a distant kraal with an artificially fed infant who had not improved as well as we had hoped. Had we been able to discharge him early and follow up his case outside the hospital, this death from bronchopneumonia might well have been prevented.

Case 2

A 8-week-old African male was admitted on 10 May 1954. He was a first-born child, his birth was normal and he was breast fed. His birth weight was unknown. Both parents were pure Africans. The infant was well until he was 5 days old when he began vomiting a little after each feed. The vomiting was not projectile in type from the beginning, but had been so for the past 5 weeks. Stools were passed normally for some time after birth, but lately there had been marked constipation and the mother said many days would go by without a stool being passed, and then only a very small quantity would be passed.

His weight was 7 lb. 5 oz. Mild dehydration was present, but there was evidence of marked weight loss. The infant was alert with a lusty cry. All systems were normal apart from the gastro-intestinal system. The abdomen was not distended. He was given a breast feed, and after this peristaltic waves were seen moving from left to right, and an easily palpable tumour was detected on the right side just below the costal margin. The surgeon consulted agreed on the diagnosis. The administration of intravenous fluids were started, and the stomach was washed out and left empty. Operation was performed and the diagnosis was confirmed. The infant made an uninterrupted recovery and was discharged 8 days after operation weighing 8 lb. 6 oz.

Case 3

A 4-week-old African male was admitted on 27 December 1956. He was a first-born child, his birth was normal and he was breast fed. His birth weight was unknown. Both parents were pure Africans. The infant was well until he was 5 days old, when he started vomiting after feeds. The mother described the vomiting as 'shooting out'. The infant never passed a stool unless given an enema.

His weight was 6 lb. 5 oz. He was thin and there was evidence of some dehydration. All systems were normal apart from the gastro-intestinal system. The abdomen was soft. Visible peristalsis was present. It was thought that a pyloric tumour was palpable. Rectal examination revealed a trace of green formed stool. This was the state of affairs on admission in the evening. Throughout the night frequent small feeds of Hartman's solution were given. The following morning, 12 hours later, it was reported that the infant had vomited each feed of Hartman's solution. No stool had been passed. Mild dehydration was still present. A bottle of Hartman's solution was offered and while sucking this, typical peristaltic waves were seen, and now a pyloric tumour was definitely palpated, followed by projectile vomiting. Intravenous fluids were given and the stomach was emptied. Operation confirmed the diagnosis. By the fifth day after the operation the infant was fully breast fed, in excellent condition and improving. On discharge on the ninth day his weight was 7 lb. 6 oz.

SUMMARY

Three cases of congenital hypertrophic pyloric stenosis in Africans are recorded. The condition would appear to be extremely rare in this race.

Note: Since completion of this paper 2 more cases of pyloric stenosis in African infants have been diagnosed in this department.

I wish to thank Dr. S. Disler, Medical Superintendent, King Edward VIII Hospital, Durban, for permission to publish this article, and Dr. H. L. Wallace and Dr. Pauline Kleneman in whose wards these cases were examined.

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EAST LONDON CONGRESS 1959 AND THE JEWISH NEW YEAR : KONGRES TE OOS-LONDEN 1959 EN DIE JOODSE NUWEJAAR

It is much regretted that the dates chosen for the 42nd South African Medical Congress conflict with the Jewish New Year celebrations. The Organizing Committee have fully investigated the possibility of a change of dates, but it has proved to be impossible because the buildings to be used for the main Congress work are closed for the Spring holidays only from Friday 25 September to Monday 5 October inclusive.

However, the plenary sessions and the main Congress social functions have been arranged to be over by Thursday 1 October 1959, so that Jewish colleagues will feel free to return home should they wish to do so. The greatest possible hospitality will of course be extended by the local Jewish community to any colleague and his family wishing to celebrate the New Year in East London.

Elizabeth McCabe
Organizing Secretary

Dit is baie jammer dat die datums wat vir die 42ste Suid-Afrikaanse Mediese Kongres gekies is, met die Joodse Nuwejaarfeesvierings bots. Die Organiserende Komitee het die moontlikheid om die datums te wysig deeglik ondersoek maar dit blyk onmoontlik te wees want die gebou wat vir die belangrikste Kongresverrigtinge gebruik sal word is slegs van Vrydag 25 September tot Maandag 5 Oktober inkluis, vir die Lentevakansie gesluit en dus vir die Kongres beskikbaar.

Reëlings is egter getref om die voltallige sessies en die belangrikste sosiale funksies van die Kongres teen Donderdag 1 Oktober 1959 af te handel sodat Joodse kollegas vry sal wees indien hul dit verkies, om huis toe te gaan.

Aan enige kollega en sy gesin wat die Nuwejaar in Oos-Londen wil vier bied die plaaslike Joodse gemeenskap die mees gulhartige gasvryheid aan.

Elizabeth McCabe
Organiserende Sekretaresse

COLLEGE OF GENERAL PRACTITIONERS

On 30 September 1958 the following resolution was passed by the Executive Committee of the National General Practitioners Group of the Medical Association of South Africa: 'The National General Practitioners Group unanimously agreed to form Faculties of the College of General Practitioners'.

It was as a result of this resolution that the letter dated 3 October which was published on page 1028 of the issue of this *Journal* of 18 October 1958, was addressed to the Hon. Secretary of the Council of the College of General Practitioners, London. This letter intimated that the Group would sponsor the formation of a College of General Practitioners in South Africa, using the faculties offered by the College of General Practitioners in Great Britain. To this end the Group is encouraging the formation of Faculties of the College in South Africa; and, when a sufficient number of Faculties have been formed, the Group proposes to apply for the formation of a South African Council. As Dr. Ian Grant stated in his address reported in this *Journal* of 25 October 1958 (**32**, 1046) a South African Council 'would to a very large extent be autonomous . . . and would have (its) own Board of Censors'. It is the intention of the National General Practitioners Group, when the South African College is strong enough, to withdraw and leave it to manage its own affairs.

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VAN DIE REDAKSIE : EDITORIAL

VERKOUE

Virus-infeksie van die boonste lugweë is een van die belangrikste oorsake vir afwesigheid van werk en korttermynongeskiktheid by die werkende bevolking van die wêreld. Die gewone verkoue maak 'n groot persentasie van hierdie virus-infeksie uit. Vanweë die eendersheid van simptomatologie is daar so 'n groot oorvleueling in die kliniese beelde van griep, verkoue (coryza), akute trageo-laringitis en ander adenovirus-infeksies, dat selfs die mees ervare klinikus die sporadiese geval met moeite in 'n waterdigte kompartement kan plaas. Gelukkig maak dit, in ons huidige staat van kennis van die probleem, ook nie veel saak nie, want die prognose en behandeling word geensins hierdeur geraak nie.[1]

'n Verkoue is by uitstek 'n sosiale siekte. Selfs jou bitterste vyand geniet jou innigste simpatie as die tranende oë, geswolle neusslymvlies en profuse rhinorree met onmiskenbare tekens van trageo-laringitis, jou herinner aan jou vorige ondervinding met ‚hierdie' virus. Nou moet ons egter 'n oomblik stilstaan by ‚hierdie' virus. Volgens C. H. Andrewes,[2] wat van 11½ jaar ondervinding in hierdie studierigting spreek, is daar ernstige besware teen die konsep van ‚'n' verkoue-virus. Daar is byvoorbeeld gevind dat sommige verkoues 'n langer inkubasieperiode het as andere, nl. 4 dae in plaas van die gewone 2-3 dae. Die vorige verkouevirus (in 1953 geïsoleer en daarna weer verloor[1]) kon maklik deur eter geïnaktiveer word terwyl daar onlangs eter-weerstandige virusse in verkouelyers gevind is. Waarskynlik is daar veelvuldige faktore wat bydra tot die ontstaan van verkoues en veral Amerikaanse werkers het die JH en 2060 virusse tydelik as van belang beskou.

Die eksperimentele werk oor verkoues word met menslike vrywilligers gedoen. In die ondervinding van werkers by die Navorsingstasie vir Verkoues te Salisbury in die Suide van Engeland, gaan die meeste verkoues nie met koors gepaard nie, en as die nuttigste kriterium word beskou die aantal papiersakdoeke per dag gebruik.[2] In hulle studies vind hulle dit ook uiters moeilik om 'n verkoue te laat ‚vat'. So bv. het 10 persone met ‚gekweekte' verkoues geen sukses gehad in die transmissie van hulle siekte nie, totdat 'n nuwe aankomeling met 'n ‚wilde' verkoue opgedaag het en in 'n ommesientjie 3 studente aangesteek het. In hierdie studiekamp word deur noue kontak 'n kruisinfeksie-syfer van 10% gevind. Verdere studies toon ook dat die virus maklik geïnaktiveer word deur uitdroging en dat droë besmette sakdoeke nie maklik die siekte versprei nie.

Die algemene opvatting dat verkoues deur ‚koue vat' veroorsaak word is waarskynlik 'n verwarring van oorsaak en gevolg. Meer waarskynlik is die koue gevoel by blootstelling aan 'n trek die eerste simptoom en *nie* die oorsaak van die verkoue nie. Dowling, Jackson en Inouye[3] toon aan dat vroue in 'n sekere fase van die menstruele siklus wel verkoues kan opdoen deur afkoeling in bv. 'n trek.

Deur al die literatuur oor hierdie onderwerp loop die bekende probleem: Wanneer is 'n toestand 'n swaar verkoue of 'n ligte griep? Dit maak nogal groot verskil aan die konsep en studie van die epidemiologie van die virus-infeksies van die boonste lugweë. Die Miksovirusse, wat Influenza A, B, C, Sendai en C.A. (kroep-verbonde) virusse insluit, kan in embrioniese weefsel van eiers gekweek word terwyl die adenovirusse, sjimpansee-virus wat verkoues in hierdie diere veroorsaak, maar waarskynlik nie in mense nie), 2060, JH en ‚die' verkoue-virusse, hoofsaaklik slegs in weefselkulture gekweek kan word.[1]

Die verdeling van die verskillende kliniese beelde is miskien kunsmatig sover dit ons as klinici raak, en die benaming is meer gemik op die anatomiese setel wat hoofsaaklik aangetas is. Dit is miskien beter om die beelde saam te vat as akute respiratoriese siektes en dan die vier hoofsindrome nl. griep, verkoue, atipiese pneumonie en katarrale ontsteking met koors te probeer onderskei na gelang van die deel van die asemhalingsweë wat die swaarste aangetas is.[1]

Influenza tas die konjunktiewe veral aan. Servikale kliervergroting is seldsaam en gering. Influenza A en B kan nie klinies onderskei word nie en albei kan longkomplikasies gee. Stafilokokkale infeksies ontstaan dikwels deur 'n sekondêre organisme.

Verkoues gaan gewoonlik nie met koors gepaard nie, en, indien teenwoordig, is daar geringe faringitis en laringitis. Sommige persone kry gereeld sekere komplikasies, soos otitis media, sinusitis of brongitis na verkoues, en dit dui waarskynlik op 'n konstitusionele faktor. Infeksies met herpes simplex virus kan as deel van die beeld voorkom.

Atipiese pneumonie is nog met duisterheid omgewe, hoewel ‚spesifieke' virusse geïsoleer is, maar die moontlikheid word genoem dat dit, in 'n sekere persentasie gevalle, mag volg op gewone verkoues as gevolg van aspirasie of meganiese faktore.[1]

Katarale toestande, deur die Engelse werkers as ‚febrile catarrh' en deur Amerikaners as ‚acute respiratory disease' (A.R.D.) beskryf, is waarskynlik die gevolg van besmetting met adenovirusse. Die keel is gewoonlik baie rooi, servikale limfadenopatie (50% gevalle) en 'n eksudaat kan in die keel aanwesig wees. Dikwels is daar 'n mengsel van die vorige beelde.[1]

Ons moet onthou dat 'n verkoue-tipe beeld as prodromale stadium van poliomiëlitis kan voorkom en difterie, hemolitiese streptokokkale infeksies, Vincent se Angina en infektiewe mononukleose, soms moeilikheid by die differensiële diagnose kan veroorsaak. 'n Pasiënt met verkoue moet tog dopgehou word vir die ontwikkeling van komplikasies omdat sy weerstand as geheel verlaag is, veral in die jonger en ouer groep—(kinders—otitis media, ou mense—pneumonie).

Hoewel ons miskien nie veel vordering gemaak het in

ons behandelingsmetodes nie, getuig 'n lewendige literatuur van groot belangstelling op hierdie gebied. Dit skyn asof die gedeeltelike oplossing waarskynlik in die terrein van die epidemiologiese metodes mag lê.[4]

Die moontlikheid van 'n outogene vaksiene as profilaktiese maatreël, word deur Ritchie in 'n onlangse artikel bespreek,[5] en in 'n ander artikel in dieselfde blad stel hy klein dosisse antibiotika voor omdat hy sekondêre infeksies belangrik ag.[6] Ons eie gevoel is dat daar waarskynlik min te kies is tussen die slagspreuk: ‚vier dae te kry, 4 dae te hou en 4 dae te verloor' of ‚twee aspirienes en 'n dop brandewyn en vroeg na bed'. Ons weet watter een die gewildste sal wees!

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CURABLE HYPERTENSION

The importance of detecting the small group of patients with *curable* hypertension hardly needs emphasizing, especially in view of the relatively unsatisfactory results of current antihypertensive therapy in essential and irreversible renal hypertension. A few conditions which may be associated with hypertension that may be amenable to treatment are discussed in this article.

Coarctation of the aorta is diagnosed by observing vigorous carotid pulsation in the neck, delay (in 95% of those palpable) or absence (in 20%) of the femoral pulses, and palpable and often visible pulsation of enlarged collateral arteries, especially between the shoulder blades.[1] The average life expectancy for adults with coarctation of the aorta is 35 years.[2] All patients should be referred for surgical correction of the constriction unless it is trivial and before the patient is too old. The optimum age for operation is between 10 and 20 years.

A history of paroxysmal headaches of an intense throbbing character associated with excessive sweating, palpitations, pallor, trembling, and a rapid rise in blood pressure, is suggestive of *Phaeochromocytoma*. This tumour may also cause persistent hypertension, and those cases in which there are no superimposed episodes of more severe hypertension are difficult to diagnose clinically. Urinary estimation of pressor substances is the most useful diagnostic test, another being the reduction of blood pressure produced by the intravenous injection of rogitine. The aggravation of the hypertension which intravenous histamine produces is not without danger and this diagnostic procedure is not recommended. False positive results occur with all these tests; they are not uncommon with the rogitine test, but are most unusual with the examination of the urine for pressor substances. Retroperitoneal air insufflation and adrenal tomography may help to localize the tumour which, however, may also occur in ectopic sites. Massage of the loin over a tumour at the bedside may precipitate a typical attack. Timely removal will result in cure, but meticulous operative and post-operative care is essential.[3] Occasionally these tumours are malignant.

Episodes of weakness, often true paralysis, associated with tetany, paraesthesiae, low serum potassium, renal potassium loss, metabolic alkalosis, and polyuria, are characteristic of *primary aldosteronism*. The adrenal gland is again the offending organ and removal of the tumour or of most of the adrenal tissue (if there is bilateral hyperplasia) should be carried out.

The obese, hirsute, polycythaemic, striated patient with *Cushing's syndrome* also has a curable hypertension, usually on an adrenal basis.

Unilateral renal disease may result in curable hypertension. *Pyelonephritis* may be the underlying cause and intravenous pyelography will show an entirely unilateral lesion. If this is the case, nephrectomy should be performed, provided the function of the remaining kidney is adequate and the diseased kidney is essentially functionless.[4] Differential clearance techniques may be of help in this regard. There is only a 19% chance of success even in the most suitable case, which is presumably due to the development of irreversible changes in the contralateral kidney resulting from hypertension. Early diagnosis is the key to cure.

Unilateral renal ischaemia (rather than infarction) may result in malignant hypertension of rapid onset and with a rapidly fatal termination if the offending kidney is not removed. Between 25 and 30 such cases have been described, the renal artery obstruction often being due to a spontaneous thrombosis of the renal artery. About one-half of these patients have abdominal pain at the time of arterial obstruction. If the history suggests this diagnosis, investigations should be exhaustive because cure is possible. Pyelography often shows little or no function on the affected side but this is not always so. Abdominal aortography may reveal obstruction of the renal artery.[5]

Many of these causes of curable hypertension can be diagnosed at the bedside or at least strongly suspected. The practitioner should be on the look-out for such cases with a view to early diagnosis and investigation and treatment at suitable centres.

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THE J. S. DU TOIT MEMORIAL FUND

The attention of members of the Association and others is called to this Fund. which has been established to perpetuate the memory of the late Dr. J. S. du Toit. The object of the Fund is to assist the sons and daughters of deceased colleagues who desire to pursue medical studies. The need for this kind of assistance has become obvious in the course of the administration of the Association's Benevolent Fund, the primary purpose of which is to prevent or relieve distress arising from financial stringency on the part of dependants of deceased colleagues. The late Dr. du Toit was one of the leading founders of the Benevolent Fund, which was always very close to his heart. He was its Hon. Treasurer

until the time of his death, and under his able and devoted guidance excellent use was made of the generosity of subscribing members of the Association. He was much interested in the idea of grants to enable the children of deceased colleagues to undertake the studies which are necessary for entry into the medical profession and for which their deceased parents had been unable to make financial provision. It is therefore highly appropriate that the name of J. S. du Toit should be associated with a fund for the realization of this worthy object.

Dr. du Toit was a member of the Medical Association for 46 years, and when he became its President in 1956 he had been Hon. Treasurer of the Association for 32 years. Apart from the special service he rendered for so long in the financial field, the influence of Dr. du Toit's personality was always felt as a strong unifying influence in the multiform membership of our Association. It is a great debt that the Association owes to him, and the promotors of the J. S. du Toit Memorial Fund appeal with confidence to their fellow members both on this ground and because of the worthiness of the object to which the Fund is to be devoted. Cheques made payable to the 'J. S. du Toit Memorial Fund' should be addressed to the Head Office of the Association, P.O. Box 643, Cape Town.

BENIGN NEOPLASMS OF THE LUNG*

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Benign tumours of the lung are rare, but their clinical recognition is of considerable importance. Clinically, all tumours of the lung must be diagnosed as malignant on statistical grounds; the diagnosis of benignity can only be established with certainty on histological examination. With benign tumours relief of incapacitating symptoms can be offered surgically with low mortality and excellent long-term results.

In my own Thoracic Surgical Unit we have seen approximately 350 cases of bronchogenic carcinoma in the past 10 years. During this period we have seen only 22 cases of benign tumour, comprised of the following pathological varieties, viz. bronchial adenoma 9 cases, hamartoma 5 cases, leiomyoma 2 cases, fibroma 2 cases, and 1 case each of papilloma of the bronchus, chondroma of the lung, endobronchial lipoma, and haemangioma. Two of these cases, viz. a leiomyoma of the right main bronchus and a fibroma of the pleura, showed histological suspicion of malignancy which, as yet, has not been confirmed by their benign clinical course.

CLASSIFICATION

These benign tumours can be classified in two ways, according either to their anatomical site or to their histological type. Whatever the pathological type of the tumour the symptoms will depend on its anatomical origin.

Anatomical Classification:
(*a*) Endobronchial (*b*) Intrapulmonary (*c*) Pleural
Pathological Classification (in order of frequency):
(*a*) Bronchial adenoma (*d*) Fibroma (*g*) Papilloma
(*b*) Hamartoma (*e*) Haemangioma (*h*) Chondroma
(*c*) Lipoma (*f*) Leiomyoma (*i*) Single cases of bronchial angioma, lymphangioma and neurofibroma have also been described, according to Langston.[1]

Most of these tumours present anatomically either endobronchially or interstitially, e.g. adenoma, hamartoma, lipoma and leiomyoma. The fibromata presents either endobronchially or from the pleura. The papillomata present only within the bronchus. Haemangiomata present only in the lung substance.

BRONCHIAL ADENOMA

In all published series the commonest benign tumour is the bronchial adenoma. There is considerable discussion whether these tumours ought strictly to be included amongst the benign lesions. The British school on the whole consider them to be benign but never recommend bronchoscopic removal; the American school on the whole consider them to be malignant. McBurney *et al.*[2] state that of their 111 bronchial adenomas 9 metastasized, and on a review of the literature they found that of 700 tumours 78 had shown clinical metastases. Goldman[3] states, 'It is unlikely that malignant change often occurs in bronchial adenoma but from its inception it is either benign or malignant'. Kincaid-Smith and Brossy[4] describe a case of bronchial adenoma in a female of 58 in whom a right middle and lower lobectomy was performed and in whom a solitary secondary of similar histological character was removed from the liver 6½ years later.

Pathogenesis and Pathology

In 1938 Womack and Graham[5] stated, 'These tumours are of mixed developmental origin arising from rests of foetal lung'. In this way they explain the tendency in these tumours to a varying histological structure. Willis[6] however, states that they arise from the mucous glands of the bronchial wall. It should be recalled that these glands lie partly outside the cartilages and partly superficial to them in the submucosa. That is why the bronchial adenoma is partly submucosal and partly outside the cartilage ring. The important feature is that, even if a small portion of the bronchial adenoma is visible bronchoscopically, it is often like an iceberg with 9/10ths of the tumour outside the bronchial wall.

Liebow[7] states that there are 2 types of bronchial adenoma, as follows: (1) The carcinoid type comprises 85% of the cases of bronchial adenoma. Columns or groups of cells are seen, separated by highly vascular stroma, and a pseudo-acinar picture may be simulated. The cells are regular both in size and in staining property. This histological group gives the best prognosis. (2) Cylindroma consist of branching, tubular epithelial structures with irregular acini and are more invasive microscopically, with a tendency to mucin formation. They account for about 15% of the bronchial adenomas.

Further subdivision of bronchial adenoma is in my opinion not justified.

Site. Most bronchial adenomas occur in the visible bronchi, the right middle and lower bronchi being common sites.

Age. Most of these tumours occur between the ages of 30 and 40. Sherman[8] in 1956 stated that there had been only 10 cases of bronchial adenoma under the age of 14; Ward[9]

* A paper presented at the South African Medical Congress, Durban, September 1957.

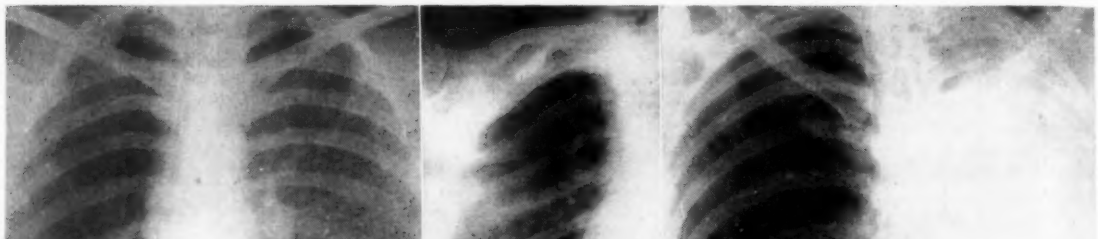

Fig. 1. Note the emphysema of the right lung field as shown by the sparse lung markings compared to those on the left. A bronchial adenoma of the intermediate bronchus has produced atelectasis of the right middle and lower lobes with compensatory emphysema of the right upper lobe.
Fig. 2. Lordotic film showing right middle lobar atelectasis due to bronchial adenoma in a female of 22 years.
Fig. 3. Radiograph demonstrating left-sided pneumonic atelectasis due to bronchial adenoma in a female of 39 years.

reported the youngest in a child of 7 years. We have had an adenoma in a boy of 12, who remains well 8 years after right middle and lower lobectomy.

Sex. Most cases are described in females. Of the 9 cases we have seen, only 2 occurred in males.

Symptoms

In most cases the presence of bronchial adenoma causes definite symptoms, but Good[10] reports that of 100 consecutive cases 17 were quite asymptomatic and were found incidentally on radiological investigation. The symptoms depend on the site of the tumour, whether obstruction to the bronchus is complete or incomplete, and whether infection has occurred distal to the obstruction.

(*a*) *Symptoms due to the tumour:*
(i) Ulceration of the tumour frequently occurs with resulting haemoptysis. This occurred in 6 of our 9 cases.
(ii) A dry troublesome cough due to the presence of the tumour from irritation similar to that caused by a foreign body.
(*b*) *Symptoms due to obstruction:*
(i) *Incomplete obstruction* causes unilateral wheezing from the fact that air is able to enter the bronchus during inspiration but is trapped in the lung during expiration. This obstructive unilobar or pneumonic emphysema can cause marked breathlessness.
(ii) *Complete obstruction.* Sudden atelectasis may cause pleuritic pain and discomfort from the negative pleural pressure. This atelectasis is often accompanied at first by severe breathlessness.
(*c*) *Presence of infection:*
(i) Suppuration occurs distal to the obstruction and a productive cough results.
(ii) Intermittent obstruction results in an abscess distal to the obstruction and produces intermittent expectoration of pus and all its sequelae.
(iii) Infection in an atelectatic lobe may result in bronchiectasis with continual cough, sputum, haemoptysis and pyrexia.
(iv) Infection sometimes spreads to the pleura, causing pleuritis with effusion. Empyema may result, with the development of a broncho-pleural fistula. We have had two such cases which were drained for many months and continued to suffer severe haemoptysis and produce pus. One of these was in a female with a bronchial adenoma and the other in a male with a papilloma of the bronchus. Both of them were treated by extrapleural pneumonectomy and pleurectomy.

The most important symptoms, therefore, are those of haemoptysis, cough, repeated lower respiratory infection and unilateral wheeze.

Radiological investigations:

The diagnostic X-ray appearances of bronchial adenoma are as follows:

(*a*) In 20% of cases the tumour mass itself is seen as the only radiological abnormality on X-ray.

(*b*) By far the larger number of cases are shown on the X-ray by the secondary effects of the tumour, as follows:

(i) Emphysema due to partial obstruction will be seen on screening or on comparing the films of inspiration and expiration.

(ii) Atelectasis will be seen when obstruction is complete. It may be either segmental, lobar (Figs. 1 and 2) or pneumonic. (Fig. 3.)

(iii) The presence of infection will be shown by an abscess, multiple abscess cavities, bronchiectasis, empyema, or broncho-pleural fistula.

A bronchogram, in my opinion, has little place in the diagnosis of the lesion, for it does not supply any pathological confirmation.

Bronchoscopy

This is the most valuable adjunct to diagnosis, for over 98% of these tumours occur in the visible bronchi. The site of the obstruction will be confirmed and pathological examination will be established.

Treatment

Bronchoscopic removal, so ably introduced by Chevalier Jackson, has few supporters today and is roundly condemned by most. McBurney[11] in 1952, stated, 'Since 1948 at the Mayo Clinic no cases have been treated by bronchoscopy as definitive treatment'. Irradiation is also of no value. Treatment is essentially surgical. Ideally, if the tumour is in a main or lobar bronchus and has not yet caused any secondary suppuration distal to it, bronchotomy is the treatment of choice. This was carried out in 1947 by Sir Clement Price Thomas.[12] Thoracotomy is performed, the tumour palpated through the posterior wall of the bronchus, which is opened, and the tumour with a sufficient sleeve of bronchial wall is excised and the bronchus reconstituted. This procedure is of value because it is conservative, without sacrifice of lung tissue. It has not been suitable for any of our cases; in all of them pulmonary resection has been necessary either because of the extent of the lesion or because of gross secondary infection. Seven of our cases came to surgery; in 3 of them pneumo-

nectomy was performed, and in 4 right middle and lower lobectomies.

HAMARTOMA

The term hamartoma was coined in 1904 by Albrecht,[13] who defined it as 'comprising a tumour-like malformation in which occur only an abnormal mixing of the normal components of an organ. The abnormality may take the form of a change in quantity, arrangement or degree of differentiation, or may comprise all three'. Histologically they originate in abnormal mixing of the normal structures.

Pathologically, hamartoma must be differentiated from endobronchial ecchondromas, described by Davidson,[14] which arise from the endobronchial cartilages, are covered by normal bronchial epithelium, and contain no other bronchial-wall elements.

There are 2 types of hamartoma, as follows:

(*a*) *Endobronchial.* These are rare. Donoghue *et al.*,[15] describing unusual bronchial tumours stated that 'of 11,626 patients bronchoscoped in the Mayo Clinic in 10 years there were 5 endobronchial hamartomas'. Paterson,[16] in 1956, reports the 31st case of endobronchial hamartoma. These tumours can cause all the symptoms of bronchial obstruction, though haemoptysis rarely occurs.

(*b*) *Interstitial.* These occur in the lung parenchyma and are usually found accidentally on radiological investigation. In rare cases they impinge on a bronchus, causing obstruction, and even, if they are exceptionally large, cause breathlessness. They are said to occur 3 times more commonly amongst males and have been described from youth to old age. Jones[17] described a rare case in a newborn infant.

The interstitial hamartomata are usually small, varying from a few millimetres to about 4 cm. in diameter, though larger tumours have been described. They are almost invariably subpleural in position and in rare cases may even lie free in the pleural space, being attached to the pleura only by a small pedicle. They are absolutely homogenous, firm to stony hard, and are usually lobulated and encapsulated. They are sharply demarcated from the lung and can usually be shelled out from the lung substance with ease.

Microscopically the bulk of the tumour consists of cartilage, but an abnormal mixture of the elements normally encountered in the bronchial wall are found, viz. ciliated epithelium, glandular epithelium, connective tissue, muscle, fat, and lymphoid tissues. The free surfaces of the lobules are covered by epithelium indistinguishable from the bronchial epithelium which dips down between the lobules in the form of deep clefts.

It is doubtful whether malignant changes have ever been convincingly described in these hamartomata. Simon,[18] however, in discussing a case suggests that certain histological appearances may indicate malignancy.

Incidence. Of 57 benign lung tumours described by Sir Clement Price Thomas[19] in 1954 10 were hamartomata. Rubin[20] found 28 cases in 8,000 routine autopsies—an incidence of 1 in 300. They were all asymptomatic. We have had 5 hamartomas—the youngest patient was a male of 22 and the oldest a female of 65. Of the 5 cases, 3 were females; 3 of the tumours were in the left upper lobe (see Fig. 4) and 2 in the apex of the right lower lobe; 3 showed myxomatous changes. Of the 5 tumours, 3 were shelled out from the lung by very easy enucleation; another was in a patient who was being treated in a tuberculosis sanatorium for a tuberculoma, and as the tumour felt softish and could not be shelled out lobectomy was performed (histologically the tumour showed myxomatous change); the other was in a male of 52, who was treated by lobectomy because there were glands present, and the presumptive diagnosis was that of carcinoma the bronchus.

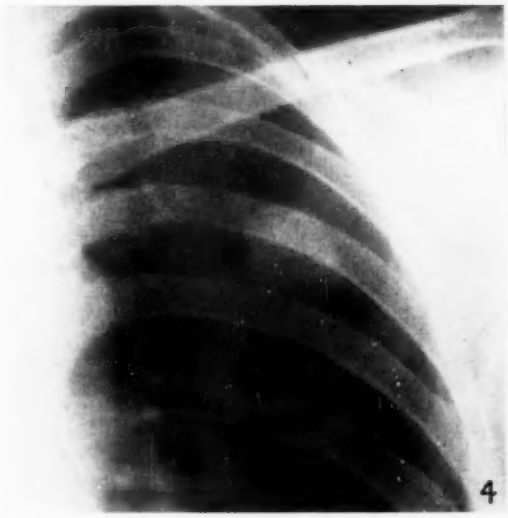

Fig. 4. Radiograph showing a well circumscribed opacity due to an hamartomain a female of 40.

FIBROMA

Fibromata of the lung must be differentiated from those arising from the mediastinum and those of neurogenic origin arising in the paravertebral gutter. Fibromata of the lung appear in two sites:

(*a*) *Endobronchial.* Of these we have no experience. Price Thomas[19] records 2 in his series of benign tumours. He states: 'They show as a lobulated mass growing within the lumen of a dilated bronchus. Histologically they show a rather cellular fibromatous tissue covered by columnar epithelium.' They present with all the symptoms of bronchial obstruction.

(*b*) *Fibromata of the Visceral Pleura.* These present such a characteristic clinical syndrome that they can often be diagnosed pre-operatively. They are sometimes silent from the chest point of view, but occasionally they present with some discomfort or pleuritic pain and a dry cough which later produces a little phlegm, but there is rarely haemoptysis. Early in their history the patients usually suffer from arthralgia, with marked clubbing and pulmonary osteo-arthropathy, which sometimes antedates the chest symptoms. The severe arthralgia disappears dramatically immediately after removal of the tumour.

Macroscopically these fibromata can be seen to arise from the visceral pleura, to which in some cases they are attached by a pedicle; in other cases they compress the underlying lung, from which they arise and from which they cannot subsequently be separated. They are hard, firm, usually smooth, occasionally lobulated, and very well defined. At thoracotomy they can usually be differentiated from malignant tumours with ease. If possible they should be removed without sacrificing lung tissue, or a thin sliver should be

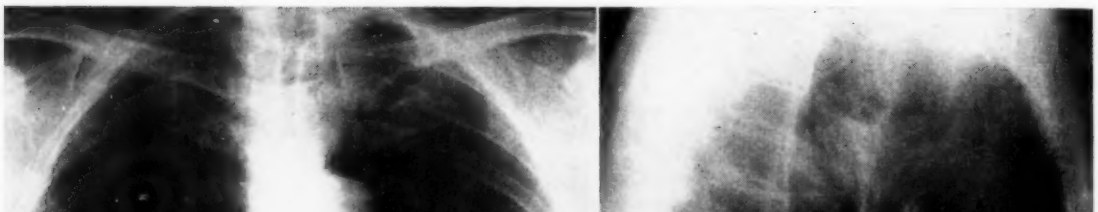

Fig. 5. Radiograph in a male of 54 years showing a dense homogenous opacity at the right base inseparable from and simulating an elevated right diaphragm or an infralobar effusion.

Fig. 6. Lateral film of the same case as in Fig. 5, showing a posteriorly situated fibroma of the visceral pleura.

removed in case there is malignant change. Often, however, the lung has been so compressed by the tumour mass that there is no plane of cleavage and lobectomy has to be performed.

Sir Clement Price Thomas[23] reviewed 6 cases of fibroma of the visceral pleura in 1953.

We have had 2 such cases, both in males. The first occurred in a man of 54 who complained of chest pain but had no clubbing of fingers. Fibroma of the pleura was suspected on radiological grounds because there was a well-defined homogeneous mass lying in the right oblique fissure and bronchoscopy was normal and bronchial washings and smears were negative for malignancy. At thoracotomy a hard, firm, well circumscribed mass arose from the visceral pleura between the two lobes, from which, however, it could not be separated, and as there was an area where the capsule appeared irregular it was felt advisable to perform a right middle and lower lobectomy. The tumour was found to be a fibroma on histological section.

Our second case was in a man of 63 who had a 6 months' history of pain and discomfort in his hands and fingers and gave a 3 months' story of a chest cold with slight productive cough. At the end of this period he consulted a doctor, who thought he had an effusion and unsuccessfully tried to aspirate his chest. He was referred for thoracic surgical opinion when an X-ray showed a huge, well circumscribed mass occupying the whole of the lower right lung field posteriorly (Figs. 5 and 6). The mass could not be distinguished either from the diaphragm or from the paravertebral gutter. Bronchoscopy showed extrinsic pressure on the right-lower-lobe bronchus, and in a Dionosil bronchogram the right lower lobe failed to fill. No evidence of malignant cells were found in the bronchial washings or bronchial slides. The pre-operative diagnosis was that of a fibroma of the pleura, which was confirmed at right thoracotomy, which showed a huge mass arising from the visceral pleura of the right lower lobe, which was grossly compressed; Mr. Denis Fuller performed an uneventful right lower lobectomy with dramatic alleviation of the patient's symptoms and early disappearance of his clubbing.

OTHER BENIGN BRONCHIAL TUMOURS

Lipoma

Intrathoracic lipomata are rare in the lung and those that have been described have mostly occurred endobronchially. Donoghue,[15] writing from an extensive experience at the Mayo Clinic, has described only 4 cases. According to Smart,[21] only 14 cases of intrathoracic lipoma are described in the literature during the period 1927-53. A 15th case treated by transpleural bronchotomy is reported by Brewin[22] in 1952. We include 1 endobronchial submucosal lipoma in our series in a man of 59. This patient had had a dry cough for 2 years, with mucus in the sputum and recurrent attacks of fever. When he was seen in consultation he was producing half a cupful of thick purulent sputum a day but never had any chest pain or haemoptysis. Bronchoscopy had shown a smooth pedunculated tumour arising distally to the left-upper-lobe bronchus. Histological examination showed an intact basal membrane with a large amount of fat underneath. Left pneumonectomy was performed by Mr. Denis Fuller because there was a markedly emphysematous lower lobe, a good deal of pus in the lower-lobe bronchi, and large glands, which fortunately proved to be inflammatory. In addition fibrocaseous tuberculosis was present, with no evidence of activity.

Haemangioma

Some authors differentiate these from arterio-venous aneurysms. Goetz *et al.*[24] state, 'It is a developmental malformation and not a tumour'. Others, however, feel that there is no differentiation between the haemangioma and the arterio-venous aneurysm.

We have had one such case in a female of 43 with exertional dyspnoea, cyanosis, clubbing, and polycythaemia. Clinically and on full investigation at the Cardiac Clinic of the Department of Medicine at the Johannesburg General Hospital the diagnosis was made of aortic stenosis, mitral stenosis, and a pulmonary arterio-venous aneurysm of the left lower lobe. Left thoracotomy was undertaken and an uneventful left lower lobectomy and mitral valvotomy performed. As the pressure gradient across the aortic valve was not sufficient to justify aortic commissurotomy the aortic lesion was not explored. The patient showed marked post-operative improvement in her cyanosis, but she collapsed 48 hours after operation with peripheral circulatory failure which could not be reversed. The immediate post-operative X-ray had suggested no abnormality but on the second day, when she collapsed, there was a suggestion of a mediastinal haematoma. At autopsy the lungs were found to be grossly haemorrhagic and oedematous, and on section showed massive intrapulmonary haemorrhages and ante-mortem thrombosis. The kidneys showed ischaemic renal tubular necrosis. Death was thought to be due to shock from the intrapulmonary haemorrhages and thrombosis, which were probably secondary to the polycythaemia.

Leiomyoma

Although muscle tumours of the lung and bronchi are extremely rare and most that are reported are sarcomatous, we have seen 2 cases of the condition. Amongst the benign tumours reported there have been 3 intrabronchial leiomyomata and 4 intrapulmonary leiomyomata. In addition 2 rhabdomyomata have been reported. Reports of only 4 surgically removed benign muscle tumours of the lung or bronchus are found.[25]

Our first case occurred in a male aged 50 who was quite asymptomatic but was included in a mass X-ray. His film showed a large, well defined, homogenous tumour mass in the right upper lobe (Fig. 7) for which thoracotomy was advised. At operation a hard, well circumscribed mass was found, occupying most of the upper lobe; no enlarged glands were found. It did not feel like a hamartoma and therefore right upper lobectomy was performed. When the tumour mass was cut across it looked like a fibroma of the uterus, and histological examination, for which I am grateful to Dr. Ian Webster of the SAIMR, confirmed that it was indeed a leiomyoma.

Our second case occurred in a 4-year-old male child seen in March 1956 with a 3 months' history of whooping cough. Following this illness the child was breathless and wheezy, and was treated for 'asthma'. During these attacks the child had high fever and scattered bilateral rhonchi were heard. Subsequently there was clinical and radiological evidence of right-lower-lobe collapse and Dr. L. B. Sunn of East London suspected a bronchial tumour. Bronchoscopy showed that the carina was broadened, and occupying the whole of the right main bronchus was a mobile, soft, friable, vascular growth, the appearances of which I thought were those of a bronchial adenoma. Dr. Webster reported that the histological features were those of a leiomyoma, and that no sarcomatous change was observed. Thoracotomy showed that the right middle and lower lobes were atelectatic, and although it might have been possible to do a right middle and lower lobectomy and perhaps remove the tumour by bronchotomy there were fleshy glands surrounding the right main bronchus, and in view of the possibility of sarcomatous change I performed a right pneumonectomy. Thus far the child has remained well apart from mild intercurrent infections.

Papilloma

Although Langston[1] states that benign endobronchial papilloma has been described, several of the papillomata that have been reported have subsequently been proved to be bronchogenic carcinoma, and the others are secondary inplants from papillomata of the larynx; the only substantiated case of primary papilloma of the bronchus is one described by Ashmore.[26] His case was that of a female aged 51 who had an haemoptysis and for whom left lower lobectomy was performed for a tumour which, on histological examination, proved to be a pure primary papilloma.

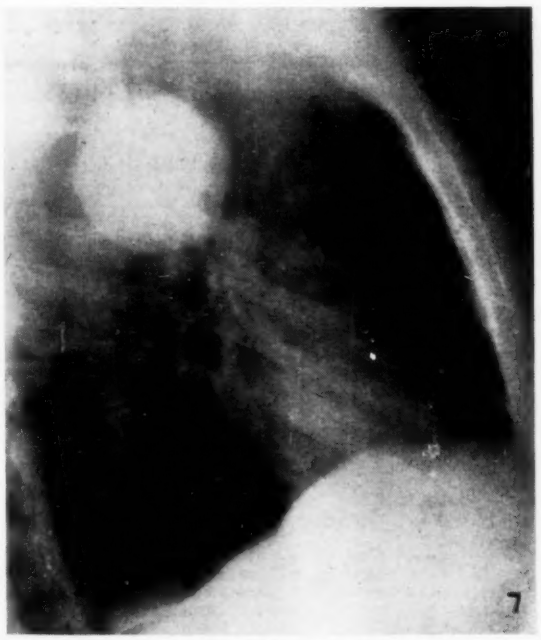

Fig. 7. Lateral film showing, in the right upper lobe of a male of 50 years, a well circumscribed opacity which could be due to any one of many causes. Right upper lobectomy showed growth which proved to be leiomyoma on histological examination.

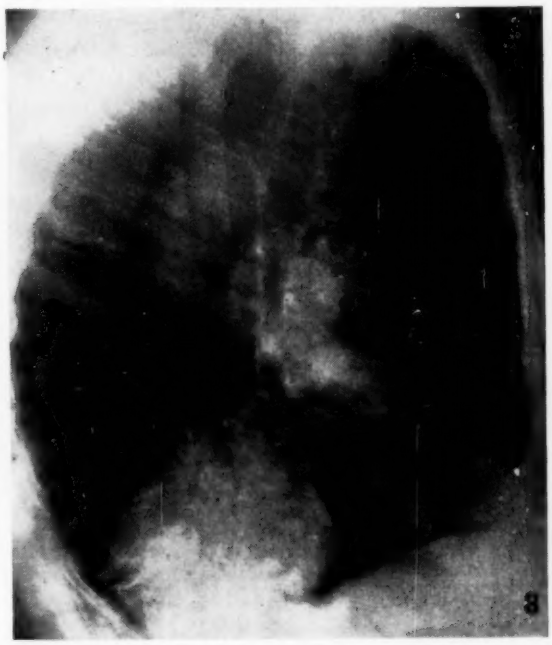

Fig. 8. Lateral film showing a well circumscribed mass with calcification. This was thought to be an hydatid, and on removal by a lobectomy proved to be a chondroma.

We have had one such case, in a male 34 years old, whom we first saw in September 1947 with a 3-year history of cough, left-sided wheeze, and subsequent pain. In 1945 he had developed an empyema for which rib resection was performed and the sinus had discharged for a year. In 1946 Mr. W. L. Phillips, after bronchoscopy, at which he had removed a portion of the tumour for section, had advised left lower lobectomy. In the year following this he continued to cough up to 2 pints of foul sputum a day and intermittent haemoptyses had occurred. An empyema with broncho-pleural fistula was diagnosed and when I bronchoscoped him in 1947 I found a sessile tumour in the left-lower-lobe bronchus. Biopsy of this confirmed the diagnosis of papilloma of the bronchus. A bronchogram unfortunately showed that he now had gross bronchiectasis of the left upper lobe as well as the lower lobe and that he had a broncho-pleural fistula. For this extra-pleural pneumonectomy was performed in September 1949 and he has remained well except for progressive exertional dyspnoea.

Chondroma

These arise from the bronchial cartilages and can be either endobronchial or parenchymal (Fig. 8). They must be distinguished from heterotopic bone formation, which is rarely seen in chronic lung abscess. We have had a pulmonary chondroma in a male of 61, whose chief symptoms were cough, discomfort in the chest, and some pleural pain. Left lower lobectomy was performed by Mr. G. Katz. A year later the patient died, and autopsy showed a squamous carcinoma at the stump of the left-lower-lobe bronchus.

SUMMARY AND CONCLUSIONS

Benign tumours of the lung are uncommon. They are of various types and either show the symptoms of bronchial obstruction or are discovered by chance on incidental radiological investigation. Bronchoscopy for pathological examination is essential if symptoms are present and, in all cases, thoracotomy, and *not* an expectant attitude, is advised, because the benign character of these tumours cannot be assured without full histological examination.

This article classifies the benign tumours of the lung, reviews present knowledge on the subject, and relates the author's experience with 22 cases of benign bronchial tumour seen in the Thoracic Surgical Unit of the Johannesburg General Hospital.

I should like to thank Dr. K. Mills, Medical Superintendent of the Johannesburg General Hospital, for allowing me access to the files of hospital patients.

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CASES OF MENINGO-ENCEPHALITIS DUE TO THE COXSACKIE A-LIKE ECHO 9 VIRUS

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During the widespread and prolonged epidemic of poliomyelitis which occurred in South Africa in 1955-56-57, a number of cases of meningo-encephalitis were investigated. Most of these cases were admitted to hospital with a provisional diagnosis of non-paralytic poliomyelitis. From many such cases poliovirus was isolated, thus confirming the correctness of the diagnosis. In many others, poliovirus was not demonstrated. From several cases of the latter group viruses, resembling Coxsackie Group A viruses in their pathogenicity to baby mice, were isolated. Of these viruses 8 were found to be serologically similar to (but not to belong to any of) the recognized serological types of Coxsackie A virus, and were therefore regarded as new types. Subsequent studies have shown that these viruses are serologically similar to Echo type-9 virus. As this virus has been responsible for widespread epidemics of infection in Europe and in Northern America, and clearly is an important cause of the aseptic meningitis syndrome, it will be of interest to note the clinical and laboratory findings in these 8 cases and to review briefly the features of the outbreaks which have occurred elsewhere.

Case 1

R.C.H., a boy aged 5 years and 11 months, was admitted to the Fever Hospital under the care of Dr. Arnold Jackson on 28 February 1956, complaining of severe headache of 4 days duration. He developed severe headache on Sunday 24 February, and asked his mother for an aspirin to relieve it. On Monday 25 February, he insisted on going to school, but did not play with his friends because of his headache. On coming home he went to bed and the family doctor was called. He suspected that the child had poliomyelitis and arranged for his admission to hospital.

On examination his temperature was 100·4°F, pulse 108/minute and his blood pressure 110/60 mm. Hg. His pupils were equal and reacted normally to light and accommodation. No abnormalities were detected in the pharynx, chest or abdomen. No neck rigidity was apparent and Kernig's sign was doubtful. Slight spasm of the hamstring muscles was evident. The cranial nerves were intact, motor power good and no sensory changes detected. The knee tendon reflexes were increased, the right ankle reflex absent, the others were present and equal. A provisional diagnosis of non-paralytic poliomyelitis was made.

The following day both knee jerks were absent. On the third day after admission the tightness of the hamstring muscles still persisted and there appeared to be slight weakness of the right quadriceps muscle.

A blood count on the day of admission showed 14·8 g. haemoglobin, 4,870,000 red cells, 6,900 white cells, of which 69·0% were neutrophil leucocytes, 3% monocytes, and 28% lymphocytes. The red cells and platelets appeared normal.

The Widal, Weil-Felix and Brucella agglutination tests, the Paul-Bunnell test, the rickettsial complement-fixation tests, and the viral complement-fixation tests for herpes, lymphocytic choriomeningitis and mumps virus infections all gave negative results.

The cerebrospinal fluid showed a cell count of 56 polymorphonuclear leucocytes and 85 lymphocytes per c.mm., a total protein of 20 mg., chlorides 677 mg. and sugar 70 mg. per 100 ml.

Specimens prepared from rectal and throat swabs were inoculated each into tissue cultures of monkey kidney cells, and a litter of baby mice. No virus was established in the baby mice, but a virus was isolated in tissue culture. This virus was not neutralized by any of the 3 type-specific poliovirus antisera and so proved not to be poliovirus. The tissue-culture fluid was then injected into another litter of baby mice, which developed paralysis, and on histological examination showed lesions resembling those produced by Coxsackie A virus.

Case 2

O.J.N., a boy aged 7, was admitted to the Johannesburg Fever Hospital under the care of Dr. Arnold Jackson on 4 July 1956 complaining of severe headache and fever which had begun the previous day.

On examination his temperature was 102°F, pulse rate 120/minute, and his blood pressure 112/78 mm. Hg. He had a flushed face with circumoral pallor. His conjunctivae were suffused. The pupils were equal and reacted equally to light and accommodation. His tongue was coated but moist, tonsils small and pharynx healthy, ears normal, and there was no enlargement of the cervical glands. No abnormality was detected in his chest and abdomen.

His cranial nerves were normal. Motor power and sensation were unimpaired. There was no neck or back stiffness, but slight tightness of the hamstring muscles. His reflexes were all present and equal.

A blood count showed 17·7 g. haemoglobin, 5,600,000 red cells, 8,700 white cells, of which 83% were neutrophil leucocytes, 2% monocytes and 15% lymphocytes. The red cells and platelets were normal in appearance.

The Widal, Weil Felix and Brucella agglutination tests, and the viral complement fixation tests for herpes, lymphocytic choriomeningitis and mumps virus, and the Paul-Bunnell test all yielded negative results.

Cerebrospinal fluid taken on the day of admission showed 129 polymorphonuclear leucocytes, 5 lymphocytes and 9 red cells per c.mm., with 40 mg. protein, 60 mg. sugar and 673 mg. chloride per 100 ml.

A virus, producing lesions in baby mice similar to those of Coxsackie Group A infections, was isolated directly from a specimen of faeces by the inoculation of a litter of baby mice.

Cases 3 *and* 4

The next 2 patients were brothers, D.H., aged 4½, and C.H., aged 2, both of whom had had 2 inoculations of poliomyelitis vaccine the previous year and had completed their course in 1956 about 2 months before the onset of their illness.

D.H. became ill on the morning of 16 October 1956 with severe headache, vomiting and fever. He was found to have slight neck stiffness and tightness of the hamstring muscles. He had no diarrhoea and no rash was noted. On the third day of illness all symptoms disappeared and he appeared quite well again.

His brother, C.H., became ill on 20 October 1956 with severe headache and vomiting, but no diarrhoea and fever. He was found to have stiffness of the neck and tightness of the hamstring muscles. His temperature continued high for 5 days, when it subsided with general improvement, though some pain in the hamstring and calf muscles of the right side still persisted. On the seventh day he was completely normal.

Both these patients were suspected of having poliomyelitis and specimens of faeces from each were sent for virus investigations. Suitably prepared suspensions were then inoculated into litters of baby mice and tissue cultures of monkey kidney cells. The mice remained healthy; a virus was isolated in the tissue cultures from each case. Baby mice inoculated with infected tissue-culture fluid became paralysed and histological examination showed a diffuse myositis similar to that produced by Coxsackie A virus.

Cases 5 *and* 6

These patients were sisters living in Durban. They were admitted to hospital as cases of non-paralytic poliomyelitis. The cerebrospinal fluid showed a pleocytosis, and from the cerebrospinal fluid of each patient a virus was isolated in tissue culture of monkey kidney cells, but not in baby mice from the original specimen. However, baby mice inoculated with infected tissue-culture fluid became paralysed and showed muscle lesions similar to those of Coxsakie A virus infections.

These viruses were isolated in the virus laboratory of the Union Health Department (under the supervision of Dr. Schapera) by Miss Westwood and Miss Hodge. The viruses were not neutralized by poliovirus antisera and therefore were sent to the laboratories of the Poliomyelitis Research Foundation for identification.

Baby mice inoculated with suspensions from the original specimens remained healthy, but a virus causing marked cytopathogenic changes was isolated in tissue culture. Infected tissue-culture fluid, inoculated into baby mice, produced paralysis and on histological examination a marked diffuse myositis, similar to that associated with Coxsackie Group A infections, was seen.

Case 7

A.P., a boy aged 8, was admitted to the Elizabeth Donkin Hospital, Port Elizabeth, under the care of Dr. Connacher, on 26 December 1956. He first became ill the day before admission complaining of headache, stiffness of the neck and back and feeling feverish. He was seen by the family medical practitioner, who suspected that he had poliomyelitis and arranged for his admission to hospital.

In the past the patient had had measles, whooping cough and chickenpox, but not scarlet fever, diphtheria or enteric fever. He had been given his first inoculation of poliomyelitis vaccine in October 1956.

On examination he was found to have a temperature of 101·2°F, pulse rate 138 and respiration rate 24/minute. No rash was seen. His throat and cervical glands were normal and no abnormalities were detected in his heart and lungs. There was marked tenderness in the right iliac fossa, but the abdomen was soft and the spleen was not palpable. The pupils were equal, there was no rigidity and Kernig's and Brudzinski's signs were negative and the reflexes were present and equal. There was no paralysis.

On lumbar puncture, the cerebrospinal fluid was under normal pressure and was clear, and was found to have 3 polymorphonuclear leucocytes per c.mm. and 10 mg. protein, 680 mg. chloride and 48 mg. sugar per 100 c.c. The Wassermann reaction was negative and no bacteria were detected either directly or in culture. The blood count showed 14·1 g. haemoglobin, 4,800,000 red cells, 11,800 white cells, of which 57% were neutrophils, 5% monocytes, 32% lymphocytes and 6% eosinophils. It was noted that there was a slight eosinophilia, but no parasites or parasitic ova were detected in an examination of a specimen of faeces.

Another specimen of faeces was sent to the laboratories of the Poliomyelitis Research Foundation for virus studies. From this specimen a virus was isolated in tissue culture, which was shown by neutralization tests not to be poliovirus. On passage to baby mice it was found to produce lesions similar to those produced by Coxsackie A virus.

Case 8

F.J.S., aged 1 year and 4 months, was admitted to the Johannesburg Fever Hospital on 24 February 1956. Six days before admission the patient began to vomit. The following day she was feverish. Three days before admission it was noted that she was unable to stand although she had walked well before the onset of her illness. She had 2 siblings both of whom were well.

On examination it was noted that she was unable to walk or stand. Her temperature was normal (98·4°F) and her pulse rate was 106. No abnormalities were found in the ears, nose or throat, or in the chest and abdomen. No rash was noted.

There was mild back stiffness and both legs were weak. The reflexes were present and equal. Two days after admission the knee and ankle reflexes of the left leg were found absent. The other reflexes were present and equal on both sides. The weakness of the left leg persisted and was still apparent on the day of discharge from hospital, 3 weeks later.

The specimen of cerebrospinal fluid which was taken on the day of admission showed the presence of 13 polymorphonuclear leucocytes and 3 lymphocytes; the total protein was 40 mg., chloride 721 mg. and sugar 56 mg.%. Bacteria were not detected on direct or cultural examination and the Wassermann reaction was negative. This fluid was inoculated into a litter of baby mice, which remained apparently healthy. Suspensions prepared respectively from a throat swab and 2 rectal swabs were also inoculated each into a litter of baby mice, but none developed

signs of illness. However, tissue-culture tubes inoculated with the rectal swab suspension showed cytopathogenic changes. In tissue-culture protection tests it was noted that this effect was not neutralized by any of the 3 types of poliovirus antiserum. Baby mice then inoculated with infected tissue-culture fluid developed paralysis. Histological sections showed lesions of the voluntary muscles resembling those produced by Coxsackie Group A virus infection.

These 8 cases presented signs and symptoms of meningo-encephalitis, including severe headache, vomiting, stiffness of the neck and tightness of the hamstring muscles and were all suspected of having poliomyelitis. The cerebrospinal fluid of the 6 cases in which this was examined, showed a pleocytosis compatible with this diagnosis. However, in none of them was poliovirus isolated.

From the cerebrospinal fluid of 2 patients, and from the faeces of the other 6, a virus was isolated. In 1 case the isolation was made directly in baby mice. In the other 7 the isolation was made in tissue culture but not in baby mice. The identity of these viruses was established in detailed laboratory studies.

LABORATORY STUDIES

Isolation of virus. Specimens of blood, cerebrospinal fluid, throat swabs and faeces were submitted from these cases for virus studies. Suspensions were prepared from the throat swabs and faeces in Hank's solution containing antibiotics. Each was then inoculated into a litter of 7 one-day-old mice and into 2 or 3 tissue-culture tubes prepared from trypsinized suspensions of monkey kidney cells.

One of the 7 baby mice of the first litter, inoculated with a suspension prepared from the faeces of case 2, developed paralysis. This one was sacrificed and a suspension prepared from its carcase and inoculated into another litter. On passage the infection gained virulence and produced evident disease in most of the baby mice (Table I). Histological sections of

TABLE I. CASE 2. LABORATORY PROTOCOL

	Baby mice	Days after Inoculation 1	2	3	4	5	6	7	8	9	10	11	12
Faeces	1	—		—	—	—	—	—	—		P*		
	2	—		—	—	—	—	—	—		—	—	—
0·1	3	—		—	—	—	—	—	—		—	—	—
	4	—		—	—	—	—	—	—		—	—	—
13 July 1956	5	—		—	—	—	—	—	—		—	—	—
	6	—		—	—	—	—	—	—		—	—	—
	7	—		—	—	—	—	—	—		—	—	—

* Kept for pathological investigation and passage, 23 July 1956.
Path. no. 5715—Limb—diffuse myositis=Coxsackie A.

		1	2	3	4	5	6	7	8
	1	—	—	—	—	W		P*	
Faeces	2	—	—	—	—	W		P	P
	3	—	—	—	—	W		P	P
Passage 1	4	—	—	—	—	W		P	P
	5	—	—	—	—	W		W	P
23 July 1956	6	—	—	—	—	W		W	W
	7	—	—	—	—	W		W	W

* Kept for passage.

		1	2	3	4	5	6	7	8	9
	1	—	—	W	W	W*				
Faeces	2	—	—	W	W	W		W	W	P
	3	—	—	W	W	—		W	W	W
Passage 2	4	—	—	W	W	—		W	W	W
	5	—	—	W	W	—		W	W	W
30 July 1956	6	—	—	W	W	—		W	W	W
	7	—	—	W	W	—		W	W	W

* Kept for passage.

		1	2	3	4	5	6	7
	1	—	—	—	—	P*		
Faeces	2	—	—	—	—	—		P
	3	—	—	—	—	—		P
Passage 3	4	—	—	—	—	—		P
	5	—	—	—	—	—		P
6 August 1956	6	—	—	—	—	—		—
	7	—	—	—	—	—		—

* Kept for passage.

		1	2	3	4	
	1	—	—	W	D	
Faeces	2	—	—	W	D	
	3	—	—	W	D	
Passage 4	4	—	—	—	D	
	5	—	—	—	P	
13 August 1956	6	—	—	—	P	Kept 17 August 1956.
	7	—	—	—	P	

P=paralysed. D=dead. W=weak.

The virus so isolated was not neutralized by Coxsackie Group A antisera A1-12 and 14-19 (A 13 was not tested) but was fully neutralized by Echo 9 antiserum.

the tissues and organs of the mice with paralysis showed an extensive acute diffuse myositis similar to that produced by Coxsackie Group A virus infections.

The litters of baby mice inoculated with suspensions prepared from the faeces of the 5 other cases showed no apparent signs of infection. Histological sections were not prepared from these mice: so it is not known whether any developed lesions of the muscles suggestive of Coxsackie A virus infections. The tissue-culture tubes inoculated with these suspensions showed cytopathogenic changes similar to those produced by poliovirus, but evolving more slowly and less completely.

The tissue-culture tubes inoculated with cerebrospinal fluid of the 2 Durban cases showed similar changes, and in passage the cytopathogenic effect was maintained.

Each of these 7 viruses in a challenge dose of about 100 TCD_{50} was then tested against each of the 3 types of poliovirus

TABLE II. TISSUE CULTURE NEUTRALIZATION TEST USING POLIOVIRUS TYPES 1, 2 AND 3 SPECIFIC ANTISERA

Virus	Virus only	Antiserum Type 1		Type 2		Type 3		Interpretation
C.C.H.	+	+	+	+	+	+	+	Not poliovirus
D.H.	+	+	+	+	+	+	+	" "
B.H.	+	+	+	+	+	+	+	" "
A.R.	+	+	+	+	+	+	+	" "
J.R.	+	+	+	+	+	+	+	" "
A.P.	+	+	+	+	+	+	+	" "
O.J.N.	+	+	+	+	+	+	+	" "

antiserum. None of the viruses were neutralized and it was concluded that none were poliovirus (Table II).

Suspensions of each virus derived from tissue cultures were then inoculated into a litter of 1-day-old baby mice. In each case a proportion of the litter developed paralysis. Histological sections showed lesions of the voluntary muscles resembling those produced by Coxsackie Group A virus.

In a series of baby-mouse protection tests, 5 of these viruses

TABLE III. BABY MOUSE NEUTRALIZATION TESTS USING A1-A19 COXSACKIE AND ECHO 9 SPECIFIC ANTISERA

Antiserum		Virus F.S.	B.H.	O.J.N.	J.R.	A.P.
Coxsackie: A	1	—	—	—	—	—
	2	—	—	—	—	—
	3	—	—	—	—	—
	4	—	—	—	—	—
	5	—	—	—	—	—
	6	—	—	—	—	—
	7	—	—	—	—	—
	8	—	—	—	—	—
	9	—	—	—	—	—
	10	—	—	—	—	—
	11	—	—	—	—	—
	12	—	—	—	—	—
	13	—	0	0	—	0
	14	—	—	—	—	—
	15	—	—	—	—	—
	16	—	—	—	—	—
	17	—	—	—	—	—
	18	—	—	—	—	—
	19	—	—	—	—	—
Echo 9 (Dalldorf)		—	0	+	—	+

0=not tested. +=protective. —=non-protective.

were tested against specific antiserum prepared against Dalldorf's classical Coxsackie A 1-12 and A 14-19 strains. Two were also tested against Coxsackie A 13 antiserum. None of the viruses were neutralized in these tests (Table III).

An antiserum was then prepared in mice against strain F.S., isolated from case 8 of this series. This serum protected against the homologous virus and against each of the viruses isolated from these patients, but not against the Coxsackie Group A 1-19 viruses (Table IV). It was concluded that these

TABLE IV. BABY MOUSE NEUTRALIZATION TESTS USING F.S. SPECIFIC ANTISERUM

Antiserum	*Virus*								
	Coxsackie A1-A19	*C.C.H.*	*D.H.*	*B.H.*	*A.R.*	*J.R.*	*A.P.*	*O.J.N.*	*Echo 9*
F.S.	—	+	+	+	+	+	+	+	+

— = non protective. + = protective.

viruses were immunologically similar and that they were representative of a new type of Coxsackie Group A virus.

When the characteristics of the Echo type-9 virus, responsible for widespread epidemics of aseptic meningitis in Europe and North America, became known, antiserum protective against this strain was received first from Dr. G. Dalldorf and then from Dr. A. Sabin and Dr. J. L. Melnick. This antiserum was tested against 4 of the 8 viruses and it was found to be fully protective (Table III). Antiserum prepared against the F.S. strain was conversely found to protect against Echo 9 virus (Table IV).

The results of this series of protection tests have revealed that the viruses from these 8 cases are immunologically similar to one another and also serologically similar to the Echo type-9 virus.

DISCUSSION

In 2 of these cases the virus was isolated from the cerebrospinal fluid and thus their role in causing the patients' illness is clear. In the remaining 6 cases the isolations were made from the faeces; thus their role was not proved. However, as the clinical pictures of the cases were similar and other viruses were not detected, there is good reason for suspecting them also as the cause of the patients' illness.

Echo virus type 9 has recently been incriminated in Europe and North America as the cause of epidemics of illness often associated with a rash, and in many cases with the aseptic meningitis syndrome.

One of the first of such outbreaks was described by Archetti and his co-workers[1] as Marche meningo-neuraxitis in Italy in 1955. From several cases a virus was isolated which produced lesions in baby mice similar to those caused by Coxsackie Group A virus infections. Subsequent studies have shown that this virus is immunologically related to, if not identical with Echo virus type 9.

Another similar outbreak, described by Garnett *et al.*,[2] occurred in Suffolk, England, in September 1955. The prominent signs and symptoms were fever, headache, nausea and vomiting, pain in the neck and shoulders, flushed face, photophobia and, in about 25% of cases, mostly children, a maculo-papular rash somewhat resembling that of measles. The cerebrospinal fluid (when examined) showed a pleocytosis. Recovery was usually rapid and complete and without paralysis or residual effect. A virus related to Echo virus type 9 was isolated from the cerebrospinal fluid of representative cases.

The following year, 1956, Rotem[3] noted that from July to November 100 patients suffering from aseptic meningitis, were admitted to the Leicester Isolation Hospital. These cases had an acute onset, headache, pyrexia, vomiting, neck and back rigidity and occasionally a rubelliform rash. Pleocytosis, predominately lymphocytic, was found in the cerebrospinal fluid of most patients. All patients recovered without specific treatment and without serious sequelae. A virus related to Echo virus type 9 was isolated from faeces, cerebrospinal fluid, and throat swabs from several cases.

A similar outbreak of aseptic meningitis with exanthem occurred in Coventry in 1956 and has recently been described by Galpine and his associates.[4] Fifty-one cases were admitted to hospital. In some of the patients the history suggested a pre-meningeal phase followed by a remission before the onset of meningeal symptoms. In most there appeared to be an early onset of meningeal symptoms. The patients presented with headache (often severe), irritability, unimpaired consciousness and pyrexia. Neck and back stiffness was noted in over a half of the cases, but in 20 of the 51 these signs were slight or absent.

A rash was seen in 19 of these cases. It was first noted from the first to sixth day of illness. In 17 cases it was erythematous, in 1 mixed erythematous and petechial, and in 1 petechial. The rash consisted of small pink discrete macules or maculopapules and always involved the face. In some cases it spread to involve the neck, shoulders and trunk, and in one infant, 15 months old, became generalized.

The cerebrospinal fluid showed a pleocytosis of usually less than 500 cells per c.mm. although in 2 cases it was over 2,000. The proportion of polymorphonuclear leucocytes and lymphocytes was approximately equal. The blood count showed a normal total or a leucopenia due to a neutropenia.

From 19 of 27 patients examined, a virus related to Echo virus type 9 was isolated. In addition, 18 of the patients showed a fourfold or greater rise in antibody titre against this virus during the course of their illness.

Boissard *et al.*[5] recovered viruses from the cerebrospinal fluid, throat washings and faeces of patients in a number of outbreaks of aseptic meningitis. They noted further that a number of these strains were found to cause lesions resembling those of Coxsackie Group A infections in newborn mice.

Maclean and Melnick,[6] who also studied strains of virus isolated in England in 1955 and 1956, noted that these strains produced myositis and paralysis in infant mice indistinguishable from that produced by Coxsackie Group A viruses. Also in 1956 a widespread epidemic of this condition involved most of Western Europe and the findings in various countries have been reported. Thus von Magnus[7] in Denmark isolated 21 viruses from a total of 147 specimens of cerebrospinal fluid tested. These viruses were neutralized by Echo antiserum type 9. The first tissue culture passage of 10 strains produced paralysis in newborn mice. The remaining 11 were non-pathogenic even after 5 passages in tissue culture. In a study of acute and convalescent sera from 14 patients, an increase in the neutralizing antibody titre was noted to occur in 13 instances.

Outbreaks of similar illness in which Echo virus type 9 was incriminated as the cause, have been described in Belgium,[8] Holland[9] and Germany,[10], and also in Canada[11] and the USA.[12] This virus has thus been responsible for one of the most extensive epidemics of aseptic meningitis so far recorded.

The present study has shown that the same infection was widespread in South Africa during 1956, as cases occurred as far apart as Johannesburg, Durban and Port Elizabeth. However these cases were sporadic and during this period no epidemic of this infection was recognized. More widespread epidemics may occur in the future. For this reason the clinical findings have been described in detail and the features of the extensive epidemics which have occurred in Europe and North America have been noted.

Whether Echo virus type 9 is a Coxsackie A virus or not is an undecided question. It has all the properties which entitle it to be placed in this group. However, the primary isolation of nearly all strains has been made in tissue culture and not in baby mice. Only after passage through tissue culture have these produced obvious illness in the baby mice. It is, therefore, of interest to note that one of the South African strains was immediately pathogenic to baby mice. This suggests that there may be some variation in virulence, or in dose, determining their pathogenicity to baby mice, a variation which would not merit a distinction from other Coxsackie A viruses.

These findings emphasize that Echo and Coxsackie viruses are closely related and it has been suggested that they and the polio viruses should be grouped together as Enteroviruses.[13]

When first discovered the pathogenicity of the Echo viruses was not known. Many of them were isolated from the faeces of cases diagnosed as non-paralytic poliomyelitis or as aseptic meningitis. There was, therefore, a suspicion that they might be concerned in the aetiology of some of these cases. From the findings of the investigations reviewed in this paper it is clear that Echo virus type 9 has caused a widespread epidemic, almost a pandemic, of an illness often associated with a morbilliform rash and many cases of which developed meningo-encephalitis.

Other investigations have incriminated Echo viruses type 4 and type 6 as the cause of outbreaks of aseptic meningitis in Europe,[14] North America[15] and South Africa.[16]

Echo viruses types 2, 3, 7, 14 and 16 have also been isolated from individual cases of aseptic meningitis.[13] There is thus a suspicion that some of these types may also have a role in the aetiology of this syndrome.

It is clear that this newly discovered group of viruses includes important pathogens of man and includes some of the commonest causes of the aseptic meningitis syndrome, which has to be distinguished from non-paralytic poliomyelitis.

SUMMARY

During the epidemics of poliomyelitis which occurred in South Africa in 1955-56-57 a number of cases diagnosed as non-paralytic poliomyelitis were investigated and were found not to be due to poliovirus. Eight such cases are described. These had fever, severe headache, vomiting, often a stiff neck and occasionally a stiff back and tightness of the hamstring muscles and some alteration, usually loss, of some tendon reflexes. In none was a rash noted. The cerebrospinal fluid of 6 cases in which this was examined, showed a pleocytosis.

From the cerebrospinal fluid of 2 patients and from the faeces of the other 6 a virus was isolated. In one case the isolation was made directly in baby mice. In the other 7 the isolation was made in tissue culture, but not in baby mice. These viruses produced lesions in baby mice similar to those of Coxsackie A virus infections. They were shown to be serologically similar, but were found not to belong to any of the recognized serotypes of Coxsackie A virus and were therefore regarded as representatives of a new serotype. Subsequent study revealed that they were similar to Echo virus type 9.

As this group of cases included patients in Johannesburg, Durban and Port Elizabeth, it is apparent that this infection was widespread in South Africa at the time, but no epidemic was recognized.

Following epidemics in Italy and England in 1955, almost a pandemic of this infection occurred in the northern hemisphere in 1956 and outbreaks occurred in most countries of Europe, in Canada and in the USA. The clinical features of the illness in these epidemics are noted. The cases were characterized by fever, headache, vomiting, and in about 25% of cases by a rubelliform rash consisting of small pink macules involving the face and in some cases spreading to the neck, shoulders and trunk. Many cases developed the signs and symptoms of aseptic meningitis and a pleocytosis was found in the cerebrospinal fluid. The course of the illness was benign and the patients recovered fully.

Other investigations have incriminated Echo viruses types 4 and 6 as causing aseptic meningitis, and other types are also suspected. It is clear that the Echo viruses are important pathogens of man and amongst the most frequent causes of the aseptic meningitis syndrome.

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ACTIVITIES OF THE BRITISH COLLEGE OF GENERAL PRACTITIONERS*

IAN D. GRANT, *President*

UNDERGRADUATE EDUCATION

The function of the Undergraduate Education Committee is to advise the College on all matters concerning undergraduate education. In its most recent recommendation to medical schools, the General Medical Council states: 'It is desirable that the student should be given opportunities to learn something of the work of the general practitioner. During the study of all clinical subjects, the attention of the student should be continuously directed by his teachers to the importance of the interrelation of the physical and psychological and social aspects of disease.' The first task of the Undergraduate Committee was to ascertain in how many of our medical schools there was attempted any teaching of general

* Three addresses given by Dr. Grant during his recent tour in South Africa.

practice. In only 9 schools out of a total of 28 was there any instructions in general practice, and in only 2 of them was there a teaching unit. The old apprentice system may have had many faults but, when it was terminated, the teachers of that day were all men who had been for a long time in general practice and many of them were still working as part-time general practitioners, and students, even in hospital, were still taught quite a lot about the treatment of cases in the home. But today the student is taught by members of a hospital staff who are highly skilled specialists but who have had no personal experience of family doctoring. Many of them have never treated patients in their homes.

The College believed that a change of heart was needed in the schools and that we should develop collaboration between general practitioners and hospital doctors. We consider that all students, whether destined to become family doctors or specialists, should know more of the good general practitioner's standards and methods and thereby more accurately judge the quality and significance of his work. We believe that the time has come for medical schools and general practitioners to combine over the training of family doctors. Both should recognize their responsibility for the quality of general practice in Britain. And so was born the student attachment scheme—the Deans argued that the curriculum was full, but again and again in the past 50 years it has been shown that the curriculum can be adjusted to the needs of the student, and each year more students voluntarily enrol for one or two weeks' attachment to a selected and capable general practitioner. To date, over 1,200 doctors have agreed to take students for a week or more in their practices, and over 1,000 students have accepted their offer. By the end of 1957 all the medical schools in Britain had some formal lectures given by members of the College. All use the student attachment scheme, and 3 of the London schools have a Director of Studies in General Practice. This, I think, may be considered a very real achievement by the College.

In 1956 a conference on undergraduate education was held in London. Each Faculty sent a representative and each medical school sent, as a guest, a senior student. The students were all agreed that they would like to learn more about the work of the family doctor, but the majority felt that lectures were not sufficient and desired to have more opportunities for seeing patients in their homes and obtaining first-hand experience of the work of the general practitioner. All too often in the past the achievements of those working in hospital have tended to suggest to students that only in hospital can satisfactory medicine be practised. The attachment scheme has changed that outlook, and those students who return to hospital after 2 weeks in general practice, and more particularly in country practice where still is conducted a considerable degree of minor surgery, midwifery and even laboratory work, are now satisfied that a full and satisfying career can be provided in general practice. Before the foundation of the College only 1 student in 20 wished to enter general practice as a first choice: now 1 in 5 desires to be a general practitioner. Unfortunately for the ambitions of these students, 1 in 2 is economically forced to enter general practice.

The General Medical Council reviews the medical curriculum approximately every 10 years, and the latest review has just been completed. The policy of the Council is to interfere as little as possible with medical schools, and to leave every school as free as possible to experiment with its own curriculum; and, although it views favourably some instruction applicable to general practice, it does not demand that instruction as obligatory. We, as a young College, must appreciate that attitude, and we must consider the ways and means by which we can make our own contribution to medical education. It is, therefore, by close association at Faculty level with the medical schools that we can make our most effective contribution. Although as a College we have much to offer the undergraduate, we must realize that it is the duty of the medical schools to keep the curriculum truly basic and that it is not their responsibility to turn out ready-made general practitioners. In so far as we can be permitted to assist in the training of the student, we must bear in mind that what we have to offer should be just as relevant to the future consultant or public health officer as it is to the student who intends to make general practice his career. We do believe that each school should appoint an experienced general practitioner as adviser in general practice and that, whenever possible, a Department of General Practice should be established in each school. The Edinburgh department is an outstanding example of how efficiently such a department can be run.

Each Faculty has its own particular problems. In some schools the point of view of the general practitioner has already permeated the school and its teaching staff; whilst, at the other extreme, there are schools which appear unwilling to take advantage of the facilities offered by the local Faculty of the College. It is our task to examine all possible means of demonstrating to medical students the fact that socially, academically and professionally general practice is a satisfying career. We have a responsibility towards them not merely to introduce them to our own interests and problems but also to help them to see our work against the background of the whole profession which they are about to enter. Much can be done to further this aim by contact with local student societies, by the way in which we receive the students into our practices, and the way in which we assist the young graduate in his early formative years. I am one of those who are firmly convinced that the good general practitioner is of just as much value to the community as is the good consultant or specialist. The one is complementary to the other, and each in his own sphere is serving society in complete equality.

THE WORK OF THE POSTGRADUATE COMMITTEE

The Postgraduate Committee is perhaps the most important Committee of the College because the chief criteria for membership of the College is an undertaking to attend postgraduate classes for 20 hours each year or 11 half-day sessions in 2 years. It is therefore incumbent upon the Postgraduate Committee to ensure that the classes arranged are likely to be of interest and value to the general practitioner, as opposed to the highly specialized postgraduate training necessary for the aspirant to consultant status. That the College has been successful in its policy is proved by the steady increase in membership. There are now 22 Faculties in the United Kingdom, each corresponding roughly to the area served by a medical school. Each Faculty has its own postgraduate committee, which coordinates the facilities for the postgraduate education of general practitioners within its area, increases the facilities where necessary, but in no way supplants existing activities. Twenty years ago epoch-making advances in medical knowledge were comparatively few, and an occasional visit to a teaching hospital and the reading of one or, at most, two journals each month sufficed to keep the general practitioner *au fait*, not only with his own problems, but also with the new techniques in the specialties.

But in 1938 a therapeutic revolution occurred with the discovery of prontosil—the first of the chemotherapy drugs, and a few years later, the finding of penicillin, the first of the antibiotics. New techniques, new methods, new drugs and miracle surgical procedures all appeared with bewildering regularity so that the young graduate is quickly out of date unless he immediately embarks on postgraduate study. He has two main problems: (1) Which of the new drugs and techniques he is going to adopt for his own use, and (2) how he must keep in touch with what is happening in the specialized branches of medicine so that he will not withhold from his patients the full benefit of modern medicine, surgery and obstetrics. He must know not only what the specialist can offer his patient—he must also be sure that the patient is referred to the right specialist, thereby saving a multiplicity of visits to various hospital departments where the investigation by too many physicians may result in adding a psychoneurosis to the undiagnosed physical disease. Again, the introduction of the new antibiotics has increased rather than diminished his responsibilities. He must combat each virus or bacterium with the appropriate antibiotic, otherwise he may encourage dangerously resistant strains of bacteria. So greatly has the face of prescribing changed that it is estimated that 80% of the drugs in common use today were not discovered 25 years ago. The advent of the Welfare State in Britain with its various departments, both medical and social, dealing with the well-being of the individual, together with the progress made in preventive medicine, has increased the responsibility of the family doctor. He must now be fully cognizant with all these developments: when he graduates from the University, he is almost completely ignorant of this field of activity.

Firstly, then, we need *postgraduate training for the young graduate* who wishes to enter general practice. The Government has recognized this need by the institution of the trainee practitioner scheme, whereby a selected practitioner, conducting his practice on a high level of professional standards, is designated a trainer and is given a young assistant, wholly inexperienced in general practice, as a trainee. He receives a small grant of £150 per year

for acting as a trainer, and the remuneration of the trainee, about £1,000 per year, is paid by the Government. There has been a good deal of criticism of the scheme; some say the trainee is abused and treated as an ordinary paid assistant; others say the trainer does not bother to teach. Whilst these criticisms are valid, they apply not to the scheme but to the individuals working it. The College has given much thought to the problem and has published a booklet covering the type of training which should be given. The trainee should see what equipment is necessary for the surgery and for the emergency bag. He should be asked for constructive suggestions. He should be shown the administrative and financial side of practice. He should be put in touch with the various departments of the local authority, with the Ministry of Labour, the National Assistance Board and the many voluntary organizations that exist to help the unfortunate. He should have an opportunity by secondment to study various types of practice—that of the country doctor, the industrial medical officer and the doctor attached to a small cottage hospital (this type is getting rarer, unfortunately), and even to spend some time in local authority clinics. This type of secondment would effectively dispel the accusation that the trainee is exploited as a form of cheap labour. Another suggestion is that the trainee might spend a second year in hospital work as a junior registrar rotating for 3 months in turn to those departments where after his year in general practice he felt his knowledge was most lacking, e.g. Eyes, E.N.T., Paediatrics, Psychiatry, etc. A combination of traineeship and rotating-internship would constitute a particularly sound training for general practice, and would be of equal value to a registrar appointment for intending consultants.

Let us now consider *postgraduate education for the established practitioner*. Self-education by reading, by attending medical societies and by domiciliary consultations may go some way towards keeping him up to date, but there is, in addition, the need for some formal organized study, which the College lays down as at least 20 hours every year. The most important method of self-education is to carry out good general practice, ignoring the temptation to drift into slovenly and undisciplined habits of thought. The old tenet, which we were all taught in our student days, of careful history-taking, full and unhurried examination and careful record-taking may perhaps be a counsel of perfection, but it will stimulate our professional interest and involve critical reading of modern literature. With large lists and much of the day spent in providing placebos for trivialities, the general practitioner may well become weary in well-doing, but he should take every opportunity of meeting his consultant colleagues. Domiciliary consultations are perhaps the greatest benefit the National Health Service has provided for him. The gradual exclusion of the general practitioner from hospital has automatically tended to lower his standards, and the finest stimulus that could be given to a general practitioner, the finest method of postgraduate education, would be the appointment of a keen and able general practitioner as a full member of the hospital team. There is no greater mental stimulus than to take responsibility for, and to share in the care of, a difficult and perplexing case. The Ministry of Health will pay for general practitioners the fees and locum fees and expenses of post-graduate courses up to a maximum of 80 hours in 2 years, and when one tries to ascertain why more practitioners do not take advantage of this opportunity the main reasons given are lack of time, deputizing difficulties (good locums are hard to find) and lack of practice in sustained study.

The general policy of the College is to develop and improve the postgraduate facilities already available, and to arrange facilities in those areas where they are lacking. The importance of good administration cannot be over-stressed. The lectures should start promptly and end promptly and at the advertised time. There must be adequate time for discussion, which is often the most valuable part of the session. This is the moment when the general practitioner can mention the problems that have arisen in his own practice, and points that have not been considered in the formal lecture may be clarified in the discussion period either by the lecturer or by one of the practitioner's own colleagues. Very often a symposium with a physician, surgeon and radiologist, each giving his point of view, with a general practitioner either opening or closing the discussion, is the best method of giving instruction. Those who are arranging the course might well be advised to invite a general practitioner of experience to help them, because he can explain the general practitioner's point of view and ensure that the subject matter is approached from the angle which will help and interest the general practitioner, rather than from a highly technical specialized angle.

The Postgraduate Committee is therefore mainly advisory and policy-making. It acts in liaison and close cooperation with the Ministry of Health and Postgraduate Medical Federation centrally and with the Medical School or University locally. There should be an interchange of representation between the Faculty Committee and the Medical Schools and in this way courses can be arranged which are best suited to the needs of the general practitioners. Our Postgraduate Committee issues a monthly diary detailing information about forthcoming courses. It arranges an occasional symposium—e.g. one on Dyspepsia, which will take place immediately after our annual meeting. It has developed a series of lectures recorded on tapes and long-playing gramophone records, which have proved very popular with isolated country practitioners. These are kept in the central office and can be forwarded with the appropriate tape-recorder machine to small meetings of doctors in the country. After the lecture is over, the doctors discuss the subject matter over tea or a drink, and great appreciation has been expressed for this service. Those College members who wish to join register with the medical recording service and receive, at regular intervals, recordings by well-known lecturers on subjects likely to be of general interest. The service, except for postage, is free to all College members. For the formal postgraduate course, the most popular subjects are Cardio-vascular diseases, Dermatology, Obstetrics, Paediatrics, Chemo-therapy and the Rheumatic diseases. Sunday ward rounds are very popular and frequently not all applications to attend them can be accepted. In country districts where there is a small cottage hospital, a consultant will attend and discuss cases which are referred to him by the local practitioners. You will have your own ideas about the best type of postgraduate teaching for your members, and each Council of the College has complete autonomy to undertake its postgraduate study in the way most appropriate for its own members. As members of the College we may not always attain success in the treatment of our cases but we can ensure, by constant study, that we deserve success.

RESEARCH IN GENERAL PRACTICE

Research in the field of medicine seen by the general practitioner is potentially as valuable as observations made in a hospital ward or a University department. In general practice are found the beginning of disease, familial diathesis and the end result of the chronic degenerative diseases. There is thus presented to those of us who work in general practice a challenge to make a fuller contribution to the study of disease by investigating more fully the problems we are called upon to handle.

There was an era in the history of medicine when all research was general-practitioner research, for there were none but general practitioners to undertake it. Advances were made through the careful noting of facts observed during the routine daily round. In this way Jenner recognized the relationship between smallpox and cowpox and Witherings noted the diuretic effect of the leaf of the foxglove. But then came the era of specialization and the flame of general practice burned low, to be fanned into occasional brilliance by such men as James MacKenzie and more recently our past president, William Pickles, whose contributions on the epidemiology of disease in country practice attracted world-wide attention. The opportunities which an academic organization of general practitioners could offer for observational research were immediately appreciated by the Foundation Council of the College and in January 1953 the Research Committee was established. It was quickly apparent that there was a reawakening of interest in research work by doctors in general practice; the first task was to ascertain the extent to which family doctors were interested and then to find out where these interests lay. A Research Register was inaugurated to facilitate the introduction to one another of practitioners who share common research interests and to enable them to pool and exchange their information. A panel of experts willingly agreed to give guidance and advice whenever needed. Volunteers came in quickly and soon information from these applicants enabled them to be classified under 3 main headings:

(*a*) Individual workers with an interest in some particular subject of research who would appreciate guidance but who preferred to work alone.

(*b*) Group researchers willing to share their observations and clinical material with other practices.

(*c*) Group researchers willing to take part in centrally organized and planned studies.

The Research Register served the double purpose of enabling those with like interests to be put in touch with one another and of providing a suitable subject for research.

The Research Committee agreed that no work would be sponsored which could better be done by other organizations. Every project undertaken by the College should be one which would yield information applying to and of value to general practice. The first College-sponsored investigation was into the value of sulphonamides and antibiotics in the prevention of the complications of measles.

The necessity for maintaining interest among members of the Research Register was quickly recognized and the Research Newsletter was published, at first as a 4-page typescript sheet. During the years it has grown in size and stature and is now reaching maturity as the quarterly Journal of the College. The research workers, however, still have issued to them, at regular intervals, two less ambitious publications, *Between Ourselves*, a document giving information on research studies in plan as well as those in progress, and also a *Progress Report*, which is a summary of the Report made for Council by the Chairman of the Research Committee. Each Faculty now has its research committee and can undertake a local research project. The central Research Committee, however, reserves the right to see and approve any material which is to be published in the name of the College. There are now 557 members and associates on the Register, many of them from overseas Faculties in Australia and New Zealand, and it is of interest that the College study of Epilepsy is being assisted by the Western Australia Faculty.

These widening horizons have led us to form a central register of Research Projects; all Faculties, Councils and Colleges throughout the Commonwealth have been invited to subscribe information for the register and to suggest research projects.

An epidemic observation unit has been established whereby all members of the Research Register are alerted to watch for the occurrence of definite disease patterns of whose diagnosis we may not be quite certain.

Each year, after the annual meeting, a research symposium is held, which has proved of wide interest. Last year we discussed ways and means in general practitioner research, and this year we hope to discuss the subject of Dyspepsia.

In regard to collaboration in therapeutic trials of new substances produced by pharmaceutical firms, the Council have decided for the present that, whilst there is no objection to individual doctors so collaborating, it is perhaps unwise for the College to sponsor the trials, mainly on ethical and legal grounds. It has agreed, however, that the Research Committee shall be free to enter into discussion with any pharmaceutical firm which desires to submit a proposal for clinical trial. The Committee will study the plan and note any foreseeable difficulties and, when these are overcome, the proposed trial can be brought to the notice of members in the Research Register; but it will be left to each individual doctor to decide whether or not he contacts the firm with a view to helping in its trial. The College require that any advertising matter relating to the trial must first be submitted for approval to the Council of the College. During the past year, 7 firms have approached the College, and 2 proposed clinical trials have been circulated to our Research Register members.

Our relations with the Medical Research Council, the Nuffield Hospitals Trust, the London School of Hygiene and the General Register Office are all cordial. At the request of the last-named, an investigation into the health of males in the years of retirement has been commenced.

The undernoted College-sponsored investigations may be of interest to you in South Africa:

Chronic bronchitis—sometimes known as the English disease, for it is commoner in the British Isles than anywhere else in the world—has little known about its aetiology, prevention or treatment, which is still largely empirical. A research is being conducted into this condition, in which 120 members of the Register are taking part, and the cost of coordinating the investigation is being met by a grant of £4,500 from the Nuffield Trust.

Eighty doctors are engaged on a study of Epilepsy. Fifty doctors are studying the incidence of Diabetes in the community. Forty doctors are investigating the cause of Asthma in children. Over 60 other Faculty-sponsored investigations are taking place.

These projects need money and the College Council feel that the Research Fund must soon be greatly increased. The doctors themselves are willingly giving of their time and many indeed have found that this research activity has opened for them new horizons and given them a new interest in their daily work. But, correlating all the data which are received requires expert statistical staff and, unless the Research Committee can be assured of financial aid for the development of its plans and projects, the outstanding progress already made cannot be long sustained. The work in which we are engaged is of inestimable value both to medicine and to the general community. To our great industrialists we make this appeal: 'Give us the tools and we will do the job.'

The Research Committee has found convincing evidence that amongst their fellow general practitioners there are many who are capable of making a worthy contribution to medical knowledge. It is hoped that the academic isolation which is felt by so many general practitioners will gradually disappear and that once again the ideas which are born of observation by family doctors in their patients' homes will become a growing source of inspiration for research. If, as a result of the guidance and coordination of research by general practitioners, new knowledge is found, new treatments are established and new interests are created, then the College will indeed have served one of the main purposes for which it was founded.

IN MEMORIAM

THEOPHILUS LÖTTER, M.B., CH.B. (L'POOL), L.R.C.P. & S. (EDIN.), L.R.F.P.S. (GLAS.), D.P.H. (CAPE TOWN)

Dr. H. Nelson, Medical Officer of Health, Pretoria, writes: It is with the greatest sadness that I have to report the death of Dr. Theo Lötter, , who passed away on 15 October 1958, after a short illness of only 5 days.

Dr. Lötter was born at Pearston, Cape Province, on 31 March 1898. He matriculated at the Boy's High School, Paarl, from where he went to the University of Cape Town to do his first two years of medical studies, before proceeding overseas.

He qualified at Liverpool University and during his student years he took a very active part in sport and was an outstanding first-team rugby wing both for his school and university. At Liverpool he distinguished himself at athletics at many important interversity competitions. He was a first-class tennis player and right up to the end was a very low-handicap golfer.

He came back to South Africa in 1927 to set up practice in Douglas, Cape Province. In 1928 he married Maggie, a daughter of the well-known Blignaut family of Paarl. After some years of general practice he returned to the University of Cape Town, where he took his D.P.H. in 1936.

He was appointed Deputy Medical Officer of Health of Pretoria in October 1936, and served the city in that capacity until a few days before he died. His sudden and untimely end has come as a great shock to the whole of the municipal service. There is a feeling of tragic and irreparable loss of a kindly friend, who always had a smile and helping hand for everybody.

Dr. Lötter was a man of kind and gentle disposition. He would go far out of his way to do a good turn. He would never turn anybody away who came to him for help, whether it was during office hours or after. The public and his patients had free access to him at all times, and never left his office without a feeling of having met a man whom they could trust and in whom they could have confidence. His ever-ready friendly smile and speech would set the most agitated person at rest, who would come under the spell of the friendly atmosphere which he, more than most people, seemed to be able to create with such ease. I think it was a natural and genuine love for his fellow man which radiated from within him and permeated the atmosphere surrounding him. There was always an air of calmness and confidence at any clinic where he was in charge. He had a special love for little children and a particular knack with them. They seemed to sense a feeling of

friendliness in his presence, and went to him quite readily with the confidence which little children have in such people.

He was a man of great integrity and a gentleman to his fingertips. He loved his work and carried out his duties faithfully. It is much to be able to say of a municipal employee that over his entire 22 years service there was not a single justifiable complaint levelled against him by any member of the public. Dr. Lötter, throughout his career maintained the dignity of his profession at a very high level. He was a credit to his colleagues and his exemplary character and high standard of medical ethics will always remain as a shining example to those of us who knew him. Although a public-health official he personified the old type of family doctor for whom the world cries out today.

He was first and foremost a family man. He loved best of all to be home with his wife and children, for whom he had the greatest love and devotion. He was never happier than when he was quietly at home with them.

Yes, a kindly friend and gentleman has gone to his rest. Those of us who knew him learned to have a great affection for him. We shall miss him but shall never forget him. To his wife, his daughter, his son and all members of his family, go our heartfelt sympathies.

HENRY WOOD WIER, M.B., CH.B. (EDIN.)

The death occurred suddenly at Robben Island on 29 September 1958 of Dr. Henry Wood Wier, at the age of 71 years.

He was born in Ballymena, Northern Ireland, and was the son of Mr. John Wier, who was the proprietor and editor of the *Ballymena Observer*. He qualified at Edinburgh University in 1914. He joined the Royal Army Medical Corps at the outbreak of the first World War and served throughout in France and Salonika, being attached to the Cameron Highlanders. In 1918 he was with the British forces which were sent to Russia during the Bolshevik Revolution. After the war he held various hospital appointments in Dublin, Chester and the Royal Edinburgh Infirmary. He later joined the P. & O. Line as a ship's surgeon and served on the Britain to Australia route.

Late Dr. Wier

In 1922 he came to South Africa and started practice in Greytown, Natal. He married Miss Doris Jackson of Estcourt. In 1931 he went to Glencoe, Natal, as Railway Medical Officer. He also held the post there of Medical Officer, Northfield Colliery. In 1939 he settled in Cape Town as Railway Medical Officer, Salt River. He was on military service from 1941 to 1945 with the S.A.M.C., being stationed at Sonderwater, Saldanha Bay and Wynberg. He retired from the Salt River post in 1952. For a short while he occupied a post at the Fort England Mental Hospital in Grahamstown. In 1953 he was appointed to the position which he held until the day of his death, viz. resident Medical Officer at Robben Island.

Dr. Wier was for many years a keen bowler and was a member of the Rondebosch Bowling Club. He was also an active member of the Ulster Association.

He was predeceased by his wife earlier this year, and is survived by his three sons and one daughter.

Dr. C. J. Stoney, of Wynberg, Cape, writes: The life of Dr. Henry Wood Wier came to a close unexpectedly on the evening of 29 September on Robben Island, where he was, in the last 5 years of his semi-retirement, medical officer to the Union Defence Force. That very morning we had journeyed together on the ferry to Robben Island—a trip he always enjoyed—and he had recounted in detail, with obvious pleasure, a holiday jaunt he had just completed round the Union, dropping in on friends and relations here and there. Although I had met him occasionally during the war (he was a veteran of the two world wars) and subsequently, I really got to know him during this last year of his life.

Dr. Wier's first joy in life was, undoubtedly, his family—his daughter and his three sons—and he was devoted to his wife, Doris. Her untimely death in February of this year was a grievous blow from which he never really recovered. And then came the love of his profession and his fellow men. He was a family doctor in a very real sense, not just a healer of disease but a kind and wise counsellor—his patients' friend. Like so many men who adorned this fast-fading institution he was remarkably patient and conscientious. His interpretation of their frailties was always tempered by charity. He would consider, and deal with, the impact of illness, not only on the patient, but the family as a whole.

Dr. Wier's passing has left a void in the lives of his many friends, and, I am sure, of all the families on the Island.

Dr. L. D. Anderson, of Cape Town, writes: Dr. Henry Wood Wier was a most likeable man; his ready jovial laugh will be long remembered by his many friends. His devotion to his work on Robben Island was an object-lesson to all of us. When on the mainland for the week-end he was always ready to take the boat trip to 'The Island' (as he called it) in calm or storm, at any time of day or night, for any of his patients. The loss of his wife at the beginning of this year, after 35 years of happy married life, was a very great blow to him. After her death, his devotion to his children and their love of their father must have been a great comfort to him. He was seldom happier than when enjoying a quiet game of bowls with his friends, and he was always looking forward to the next game. To his children is extended the sincere sympathy of his many friends and colleagues in the loss of a loving father. He enjoyed good health up to the day of his death, and one cannot help feeling that the end, coming suddenly as it did, was probably the one he himself would have chosen.

FRANCIS THOMAS WALDRON, M.D. (LOND.)

Dr. G. D. Morgan of Knysna, Chairman, South-Eastern Division of the Medical Association of South Africa, writes as follows:

Dr. Francis Thomas Waldron died at his home in Mossel Bay, C.P., on 7 July 1958, aged 84 years.

He came to South Africa from England in 1902, and after a short period in Kimberley and in Somerset West he settled in Mossel Bay in 1908 and continued in practice there for 50 years. For many years he was a part-time Medical Officer of Health of Mossel Bay. He was well-known in the surrounding districts, where he used to be called in as consultant before the days of specialists, and even later. He was one of the founders of the South Eastern Division of the Cape Western Branch. He took a keen interest in the social affairs of Mossel Bay and was connected with various sporting bodies.

Dr. Waldron was the mainstay of the South-Eastern Division, and used to attend every possible meeting. We used to look to him for his advice in our affairs. We greatly miss him and remember him with gratitude for all he did for his colleagues.

To Mrs. Waldron we extend our deepest sympathy in her great loss.

DIE LIEFDADIGHEIDSFONDS : THE BENEVOLENT FUND

Met hartlike dank word die volgende skenkings gedurende die maand September 1958, erken.

Geloftekaarte ter nagedagtenis aan:

Dr. J. S. du Toit *deur* Dr. A. van der Poel, Ds. P. S. Z. Coetzee, le Roux en Keet-gesinne, Dr. L. J. Braun, Dr. G. T. van der Vyver, Mnr. en mev. M. J. de Kock, Dr. J. H. Harvey Pirie, Drs. Heymann, Javett, Brando en Senior, Dr. A. L. Agranat. Dr. P. J. Mitford Retief, Drs. W. P. en T. P. Mulligan, Dr. J. C. Coetzee, Dr. en mevr. F. Krone,

Dr. F. O. Fehrsen *deur* Dr. G. T. van der Vyver, Dr. en mevr. F. Krone.

Mnr. W. J. Rhodes *deur* Dr. D. G. Cowie.

Dr. T. A. Fuller *deur* Dr. E. G. van Hoogstraten, Ere-Mediese Personeel, Rondebosch en Mowbray-Hospitaal, Dr. Shirley Cole, Dr. R. Wolff, Drs. W. P. en T. P. Mulligan, Dr. J. A. Currie, Dr. Lennox Gordon, Mrs. U. Scott.

Mev. Wiggishoff *deur* Dr. H. H. Navid.

Dr. E. H. Walker *deur* Mediese Komitee, Provinsiale Hospitaal, Port Elizabeth.

Mnr. J. St. G. B. Nolan *deur* Dr. A. W. Spratt.

Dr. J. A. Levitt *deur* Dr. D. A. Fowler, Tak Noord-Transvaal M.V.S.A., Dr. en mev. D. Epstein, S.A. Vereniging van Spesialiste in Fisiese Medisyne, Johannesburg.

Mev. G. D. Jones-Phillipson *deur* Mnr. C. E. Espin, Bisset, Boehmke en McBlaine.

Totale bedrag ontvang van geloftekaarte £66 3s. 6d.

Dienste gelewer aan:

Dr. E. L. Dirmeik *deur* Drs. E. W. Emms, D. W. Burton, J. K. McCabe.

Mnr. S. Dirmeik *deur* Dr. F. Petersen.

Dogter van Dr. H. L. Goldblatt *deur* Dr. W. Mirkin.

Dr. D. P. Marais *deur* Drs. J. A. Currie, R. B. Watson, H. Clegg, L. v. d. Spuy en F. W. Purcell.

Prof. Dr. H. Verleger *deur* Dr. C. A. R. Schulenburg.

Dr. C. L. Kavalsky *deur* Dr. D. Friedlander.

Totale bedrag ontvang vir dienste gelewer £49 13s. 0d.

Skenkings:

Drs. W. Laurie, R. K. Beardmore, R. A. Newsom	£1	3	6
Kollektebussie van lede, Tak Wes-Kaapland ..	10	8	9
Opbrengs Alan Sichel-Gholfkompetisie	13	0	0
Totale bedrag ontvang van skenkings	£24	12	3

Groottotaal £140 8s. 9d.

PASSING EVENTS : IN DIE VERBYGAAN

Research Forum, University of Cape Town. The next meeting of Research Forum will be held on Wednesday 5 November 1958 at 12 noon in the large A-floor lecture theatre, Groote Schuur Hospital, Cape Town. Dr. W. P. U. Jackson will speak on 'Hypercalciuria with special reference to sarcoidosis and the action of Vitamin D'. All interested are invited to attend.

* * *

Dr. Len Stein, B.Sc. (Hons.), M.B., B.Ch. (Rand), F.R.C.S. (Edin.), het sy voltydse betrekking in die hospitaaldiens van die Provinsiale Administrasie neergelê en praktiseer nou privaat as Spesialis-Chirurg te Listergebou 140, Jeppestraat, Johannesburg. Telefone: Kamers 23-7803, woning 41-2231, noodoproepe 22-4191. Hierdie nommers verskyn nie in die huidige telefoongids nie.

Mr. Len Stein, B.Sc. (Hons.), M.B., B.Ch. (Rand), F.R.C.S. (Edin.), having relinquished his post in the full-time hospital service of the Provincial Administration has commenced private practice as a Specialist Surgeon at 140 Lister Building, Jeppe Street, Johannesburg. Telephones: Rooms 23-7803, residence 41-2231, emergency 22-4191. These numbers do not appear in the present Telephone Directory.

* * *

Addresses on Cardiac Arrest and Its Management and Resuscitation of the Collapsed Patient will be given by Mr. C. N. Barnard, M.D., M.S., Ph.D. and Dr. D. M. Jowell, M.B., Ch.B., D.A., in the Physiology Lecture Theatre, Medical School, Observatory, Cape, on Tuesday 4 November at 8.15 p.m. Films will be shown. The meeting has been organized by the Cape Town Sub-group of the Ophthalmological Society of South Africa. All medical practitioners are welcome.

* * *

Bibles in Waiting Rooms. Gideons International in South Africa provide free English or Afrikaans Bibles for placing in medical waiting rooms. Any Bible so placed that has been removed will, upon application, gladly be replaced. Scriptures for placing in hospitals, nursing homes or convalescent homes are also available. For further particulars please write, stating your requirements in full, to The Bible Secretary, Gideons International in South Africa, P.O. Box 4855, Johannesburg.

* * *

Members are reminded that they should notify any change of address to the Secretary of the Medical Association of South Africa at P.O. Box 643, Cape Town as well as to the Registrar of the South African Medical and Dental Council, P.O. Box 205, Pretoria.

Failure to advise the Association can only result in non-delivery of the *Journal*. This applies to members proceeding overseas as well as to those who change their addresses within the Union.

* * *

The Norman McAlister Gregg Prize of the Ophthalmological Society of New South Wales consisting of 100 guineas (Aust.) and a Bronze Medallion will be awarded to the British subject whose submitted original work, on any subject, is deemed to be the most valuable contribution to knowledge in Ophthalmology. Intending candidates should notify the Secretary of the Society, Dr. John Hornbrook, 147 Macquarie Street, Sydney, New South Wales, Australia, at least 2 months before the closing date 31 October 1959. Regulations for the prize may be obtained from the Secretary.

* * *

Drug Treatment of Disease. Under this general heading the *British Medical Journal* is publishing a series of signed articles by distinguished contributors on the pharmacology and therapeutic uses of drugs. The object is to be of help to the practitioner faced with a constant stream of new drugs resulting from the inventive capacity of chemists and pharmacologists, many of them, differing only in 'side-chains', having varying effects. The first article in the series, by J. H. Burn, M.D., F.R.S., Professor of Pharmacology, Oxford, on 'The Antihistamine Compounds', was published in the *B.M.J.* of 4 October.

* * *

An International Society of Clinical and Experimental Hypnosis has been formed. At present it has the support of 15 countries and has its headquarters in the USA. The Provisional Chairman is Dr. Bernard B. Raginsky, M.D., Montreal, Canada. An inaugural meeting of this Society was held on 31 October at Chicago. The object of the Society is to stimulate and improve professional research, discussion and publications pertinent to the scientific study of Hypnosis. Among scientific disciplines it will encourage cooperative relations with regard to the study and application of hypnosis, and it will bring together persons using hypnosis and setting up standards for professional adequacy and training in the field. The International Society invites to membership any clinical and scientific worker in hypnosis whose qualifications meet the requirements of its constitution, viz. that he shall have a degree in Medicine or Dentistry or the Ph.D. or its equivalent in Psychology, and he shall be a member of the appropriate society for his profession, such as the Medical or Dental Association of South Africa, and be actively using hypnosis in his clinical practice or research at the time of application for membership. Any interested persons desiring further information should write to the South African Representative of the Society, viz. Dr. M. Herman, 701 Stuttafords Buildings, 63 St. Georges' Street, Cape Town.

VERENIGINGSNUUS : ASSOCIATION NEWS

NUWE AFDELING NAMAKWA : NEW NAMAQUA DIVISION

Op Saterdag, 11 Oktober 1958, het interessante verwikkelings, wat die plaaslike geneeshere betref, in Namakwaland plaasgevind.

Met steun van drs. F. H. Bakker en Bronkhorst het dr. Ivan van Niekerk as sameroeper opgetree by 'n vergadering te Nababeep-Hospitaal wat deur dertien plaaslike geneeshere en ses geneeshere van Kaapstad onder leiding van dr. P. J. M. Retief bygewoon is.

Dr. G. M. Malan van Durbanville, Voorsitter van die M.V.S.A.-

tak, Wes-Kaapland, het die Voorsitterstoel ingeneem en na 'n paar woorde van verduideliking oor die funksies van die Mediese Vereniging oorgegaan tot die vra vir voorstelle vir die naam van 'n nuwe divisie met grense oos van Van Rhynsdorp, wes van Pofadder met die Atlantiese Oseaan en die Oranjerivier as die ander Westelike en Noordelike grense, Oranjemund ook ingesluit. Die naam van die nuwe divisie sal bekend wees as die Namakwadivisie.

As Voorsitter is eenparig gekies dr. F. H. Bakker met Ondervoorsitter dr. W. A. C. Eckard en Sekretaris/Penningmeester dr. Ivan van Niekerk en die twee addisionele lede drs. J. Rossouw en H. B. Melzer.

Dr. F. H. Bakker het die Voorsitterstoel ingeneem en na 'n paar woorde van bedanking het hy dr. J. Roux aan die woord gestel, opgevolg deur drs. J. A. Currie, A. Swanepoel en C. Barnard.

Die lesings wat bogenoemde geneeshere gelewer het, was baie interessant en het groot byval gevind.

Die naweek van 10 Oktober was baie bedrywig. Vrydagaand, 10 Oktober was 'n Burgemeesters-onthaal ten huise van mnr. en mev. J. Jowell vir die besoekende dokters gereël.

Saterdagoggend het hulle die Kopermyne besigtig en Saterdagaand 'n aangename dinee by die Springbok Hotel bygewoon. Drs. G. M. Malan, P. J. M. Retief, F. H. Bakker en H. B. Melzer het elkeen 'n paar woorde gesê.

Na die dinee het mnr. Jowell, die Burgemeester, 'n paar films oor Namakwalandse blomme vertoon.

Ons is van mening dat die naweek vir almal lank in die geheue sal bly.

RAILWAY MEDICAL OFFICERS' GROUP (M.A.S.A.) : SPOORWEGDOKTERSGROEP (M.V.S.A.)

NOTICE OF ANNUAL GENERAL MEETING

Notice is hereby given that the Annual General Meeting of Members of the R.M.O. Group, will be held at Medical House, Esselen Street, Johannesburg, on Saturday 15 November at 2 p.m. sharp.

Agenda

1. Minutes of the last Annual General Meeting (circulated).
2. Matters arising out of Minutes and confirmation of Minutes.
3. Annual Report of the Hon. Secretary-Treasurer.
4. The Chairman's Report on negotiations with the Central Sick Fund Board.
5. To receive and adopt Audited Statements of Account for period 1 September 1957 to 15 October 1958.
6. Office Bearers of the Group for year 1958/1959.
7. Resolutions from Branch Groups.
8. General.

M. Cohen
Hon. Secretary-Treasurer

KENNISGEWING VAN ALGEMENE JAARVERGADERING

Kennis geskied hierby dat die Algemene Jaarvergadering van Lede van die Spoorwegdoktersgroep te Mediese Gebou, Esselenstraat, Johannesburg, op Saterdag 15 November 1958 om 2 nm. presies gehou sal word.

Agenda

1. Notule van die vorige Algemene Jaarvergadering (uitgestuur).
2. Sake voortspruitend uit die Notule en bekragtiging van Notule.
3. Jaarlikse verslag van die Eresekretaris-Tesourier.
4. Voorsitter se verslag van onderhandelinge met die Sentrale Siekefondsraad.
5. Aanbieding en aanvaarding van ge-ouditeerde Rekeningstate vir die tydperk 1 September 1957 tot 15 Oktober 1958.
6. Ampsdraers van die Groep vir die jaar 1958/1959.
7. Besluite van Tak-Groepe.
8. Algemeen.

M. Cohen
Eresekretaris-Tesourier

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

APPROVED MEDICAL AID SOCIETIES

MEDICAL AID SOCIETIES

The following list of approved medical aid societies is published for general information. Members are requested to keep this list for reference because it no longer appears in the tariff book. After each meeting of the Federal Council an up-to-date list is published in the *Journal*, including societies that have been newly approved and omitting those that have been withdrawn.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
15 October 1958

1. A.A. Mutual Medical Aid Society, P.O. Box 9595, Johannesburg.
2. Abercom Group Sick Benefit Society, P.O. Box 715, Port Elizabeth, Cape Province.
3. African Cables Medical Benefit Fund, P.O. Box 172, Vereeniging, Transvaal.
4. African Explosives Medical Aid Society, P.O. Box 1122, Johannesburg.
5. African Homes Trust Sick Fund, P.O. Box 93, Cape Town.
6. African Oxygen Limited Medical Aid Society, P.O. Box 5404, Johannesburg.
7. Afrikaanse Pers Beperk se Siekefonds, Posbus 845, Johannesburg.
8. Alex. Aitken & Carter Medical Benefit Society, P.O. Box 2636, Johannesburg.
9. Algoa Medical Aid Society, P.O. Box 369, Port Elizabeth.
10. Argus Medical Benefit Society (Cape Argus Branch), P.O. Box 56, Cape Town.

GOEDGEKEURDE MEDIESE HULPVERENIGINGS

MEDIESE HULPVERENIGINGS

Vir algemene inligting word onderstaande lys van goedgekeurde mediese hulpverenigings gepubliseer. Lede word versoek om die lys byderhand te hou want dit verskyn nie meer in die tariefboek nie. Na elke vergadering van die Federale Raad word 'n volledige lys (wat die name van pas-goedgekeurde verenigings insluit en van dié wat onttrek is weglaat) in die *Tydskrif* gepubliseer.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
15 Oktober 1958

11. Argus Medical Benefit Society (Daily News Branch), P.O. Box 1491, Durban.
12. Argus Medical Benefit Society (Star Branch), P.O. Box 1014, Johannesburg.
13. Associated Employers Medical Aid Society, P.O. Box 7462, Johannesburg.
14. A.T.I. Medical Aid Society, P.O. Box 5057, Boksburg North.
15. Atlantic Refining Company Medical Aid Society, P.O. Box 664, Cape Town.
16. Babcock and Wilcox Medical Aid Fund, P.O. Box 545, Vereeniging.
17. Bakers Ltd. European Employees' Sick Benefit Fund, P.O. Box 692, Durban.
18. Bloemfontein Municipal Employees' Medical Aid Society, P.O. Box 288, Bloemfontein.
19. Boksburg Municipal Employees' Medical Aid Fund, P.O. Box 215, Boksburg.
20. Broderick Medical Aid Society, P.O. Box 186, Vereeniging.

21. Building Societies Joint Medical Aid Fund, P.O. Box 5728, Johannesburg.
22. S. Butcher & Sons Ltd. Medical Aid Society, P.O. Box 1004, Durban.
23. Cape Portland Medical Aid Society, P.O. Box 1067, Cape Town.
24. Cape Times Medical Aid Society, P.O. Box 11, Cape Town.
25. Cape Town Municipal Employees' Association Medical Aid Society, P.O. Box 1939, Cape Town.
26. Central News Agency Ltd. Medical Benefit Society, P.O. Box 1033, Johannesburg (excluding Cape Town and suburbs, Durban municipal area, Johannesburg and Witwatersrand, and Port Elizabeth and Pretoria municipal areas).
27. Chamber of Mines Medical Aid Society, P.O. Box 809, Johannesburg.
28. Civil Service Medical Benefit Association, P.O. Box 176, Pretoria.
29. Consolidated Glassworks Limited Medical Aid and Sick Benefit Society, P.O. Box 562, Germiston.
30. Corner House Insurance Fund, P.O. Box 1056, Johannesburg.
31. Coronation Medical Aid Society, P.O. Box 1517, Durban.
32. Crookes Bros. Ltd. Medical Benefit Fund, 301 Smith Street, Durban.
33. D.F.A. Medical Benefit Society, P.O. Box 610, Kimberley.
34. Eastern Province Cement Co. Ltd. Medical Aid Society, P.O. Box 2016, Port Elizabeth.
35. E.P. Newspapers Medical Aid Society, P.O. Box 1117, Port Elizabeth.
36. Egnep Medical Aid Society, P.O. Penge, Transvaal.
37. Elwamba Medical Aid Fund, P.O. Box 42, East London.
38. Escom Cape Western Undertaking Medical Aid Society, P.O. Box 117, Cape Town.
39. Escom (N.C.U.) Medical Benefit Society, P.O. Box 30, Colenso, Natal.
40. Everite Medical Aid Society, P.O. Kliprivier, Transvaal.
41. Federated Employers' Medical Aid Society, P.O. Box 666, Johannesburg.
42. Federation of Master Printers of S.A. Medical Aid Society, P.O. Box 4465, Johannesburg.
43. Ford Medical Aid Society, P.O. Box 788, Port Elizabeth.
44. Friend Medical Aid Fund, P.O. Box 245, Bloemfontein.
45. General Mining (Associated Companies) Medical Aid Society, P.O. Box 1007, Johannesburg.
46. General Motors Medical Aid Scheme, P.O. Box 1137, Port Elizabeth.
47. Germiston Industries Medical Aid Society, 113 Pylon House, Human Street Germiston.
48. Gledhow-Chaka's Kraal Sugar Co. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
49. Greaterman's Medical Aid Society (all branches), P.O. Box 5460, Johannesburg.
50. Hollerith Medical Aid Society, P.O. Box 7018, Johannesburg.
51. Hubert Davies Johannesburg Staff Medical Aid Society, P.O. Box 1386, Johannesburg.
52. Sir J. L. Hulett & Sons Ltd. Medical Benefit Fund, P.O. Box 248, Durban.
53. Hunt, Leuchars & Hepburn Ltd. (Transvaal Staff) Medical Aid Society, P.O. Box 47, Johannesburg.
54. Irvine Chapman Medical Aid Scheme, P.O. Box 316, Vereeniging.
55. Iscor Medical Benefit Fund, P.O. Box 450, Pretoria.
56. I.W.S. Medical Aid Society, P.O. Box 6946, Johannesburg.
57. J. W. Jagger & Co. Ltd. Medical Aid Society, P.O. Box 726, Cape Town.
58. Johannesburg Board of Executors' Medical Aid Society, P.O. Box 271, Johannesburg.
59. Klerksdorp Munisipale Werknemers Siektefonds, Posbus 99, Klerksdorp.
60. K. & L. Timbers Ltd. Staff Medical Aid Fund, P.O. Box 9, Elandsfontein, Transvaal.
61. Koegas Medical Aid Society, P.O. Koegasbridge, C.P.
62. Krantzberg Mines Medical Aid Society, P.O. Box 18, Omaruru, S.W.A.
63. Kroonstad Munisipale Mediese Hulpvereniging, Posbus 302, Kroonstad.
64. G. H. Langler & Co. Ltd. Medical Aid Society, P.O. Box 3762, Johannesburg.
65. Legal and General Medical Aid Society, P.O. Box 4870, Johannesburg.
66. Mail, Times & Express Medical Aid Society, P.O. Box 1138, Johannesburg.
67. Marley Floor Tile Medical Aid Society, P.O. Box 67, Nigel.
68. L. H. Marthinusen Medical Aid Society, P.O. Box 64, Denver, Johannesburg.
69. Masonite Medical Aid Society, P.O. Box 57, Estcourt, Natal.
70. Max Engineering Medical Aid Scheme, P.O. Box 174, Vereeniging.
71. Medical Aid Society for Transvaal Teachers, P.O. Box 28, Boksburg.
72. Metal Box Company of S.A. Ltd. Medical Aid Society, P.O. Box 7752, Johannesburg.
73. Mosenthal's Staff Medical Aid Society, P.O. Box 1, Port Elizabeth.
74. Municipal Employees' Medical Aid Society (Durban), P.O. Box 625, Durban.
75. Natal Building Society Medical Aid Fund, P.O. Box 947, Durban.
76. Natal Coal Owners' (Durban Staff) Medical Aid Society, P.O. Box 281, Durban.
77. Natal Estates Sick Fund Benefit Society, P.O. Mount Edgecombe, Natal.
78. Natal Industries Medical Aid Society, P.O. Box 1300, Durban.
79. N.T.E. Staff Medical Aid Fund, P.O. Box 39, Pietermaritzburg.
80. National Industrial Credit Corporation Medical Aid Society, P.O. Box 8296, Johannesburg.
81. National Portland Medical Aid Society, P.O. Box 21, Claremont, C.P.
82. New Consolidated Gold Fields Employees' Medical Aid Fund, P.O. Box 1167, Johannesburg.
83. Northern Assurance Co. Ltd. Medical Aid Society, P.O. Box 8615, Johannesburg.
84. Northern Medical Aid Society, P.O. Box 3437, Johannesburg.
85. Northern Rhodesia Civil Servants' Medical Aid Society, P.O. Box 294, Lusaka, Northern Rhodesia.
86. Norwich Union Life Insurance Society Staff Medical and Surgical Benefit Scheme, P.O. Box 1226, Cape Town.
87. Ore & Metal Medical Aid Society, P.O. Box 3548, Johannesburg.
88. Pietermaritzburg Chamber of Industries Medical Aid Society, P.O. Box 365, Pietermaritzburg.
89. Pilkington Group European Medical Aid Society, P.O. Box 111, Springs.
90. Polliack Group Medical Aid Society, P.O. Box 3008, Johannesburg.
91. Pongola Sugar Milling Co. Ltd. Medical Benefit Fund, P.O. Box 194, Durban.
92. Post Office Medical Aid Society, P.O. Box 303, Germiston.
93. Pretoria Municipal Employees' Sick Fund, P.O. Box 408, Pretoria.
94. Pretoria News Medical Benefit Society, P.O. Box 439, Pretoria.
95. Pretoria Portland Cement Co. Ltd. No. 1 Works (Hercules) Medical Aid Society, P.O. Box 405, Pretoria.
96. Pretoria Portland Cement Co. Ltd. No. 2 Works Medical Benefit Society, P.O. Box 7, Slurry, Western Transvaal.
97. Pretoria Portland Cement Co. Ltd. No. 3 Works (Jupiter) Medical Aid Society, P.O. Box 73, Cleveland, Transvaal.
98. Pretoria Portland Cement Co. Ltd. No. 4 Works Medical Aid Society, P.O. Box 26, Orkney, district Klerksdorp.
99. Printing Industry Medical Aid Society, P.O. Box 1993, Pretoria.
100. Prudential Medical Aid Scheme, P.O. Box 1097, Johannesburg.
101. Rand Water Board Sick Fund, P.O. Box 1127, Johannesburg.
102. Randles Bros. & Hudson Ltd. (Durban) Sick Benefit Fund, P.O. Box 1046, Durban.
103. Randles Bros. & Hudson Ltd. (Johannesburg) Employees' Sick Benefit Fund, P.O. Box 2678, Johannesburg.

104. Reckitt & Colman Medical Aid Society (S.A.), P.O. Box 1097, Cape Town.
105. 'Rennie' and 'The Consolidated' Employees' Medical Aid Fund, P.O. Box 1006, Durban.
106. Reynolds Bros. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
107. E. S. & A. Robinson (Pty.) Ltd. Medical Aid Society, P.O. Box 293, Germiston.
108. Royal-Globe Medical Aid Fund, P.O. Box 317, Cape Town.
109. Safim Medical Aid Society, P.O. Box 233, Vereeniging.
110. Safmarine Medical Aid Society, P.O. Box 2171, Cape Town.
111. Safnit Mills Medical Aid Fund, P.O. Box 11, Jeppestown, Johannesburg.
112. Santam-Sanlam Siektefonds (Alle Takke), Posbus 1, Sanlamhof, K.P.
113. Schwartz, Fine, Kane & Co. Medical Aid Society, P.O. Box 5069, Johannesburg.
114. Shell Medical Aid Society (S.A.), P.O. Box 2231, Cape Town.
115. S.A. Association of Municipal Employees' (S.A.A.M.E.) Medical Aid Fund, P.O. Box 62, Pretoria.
116. S.A. Breweries Medical Aid Society, P.O. Box 1099, Johannesburg.
117. S.A.K.A.V. Sick Benefit Fund, P.O. Box 33, Paarl.
118. S.A. Mutual Fire & General Insurance Co. Ltd. Staff Medical Aid Fund, P.O. Box 516, Johannesburg.
119. S.A. Mutual Life Assurance Society Staff Medical Aid Fund, P.O. Box 66, Cape Town.
120. S.A. Press Association Medical Aid Society, P.O. Box 7766, Johannesburg.
121. S.A. Teachers' Association Medical Aid Society, 12 Bellevue Road, Sea Point, C.P.
122. S.A. Torbanite (Boksburg) Medical Aid Society, P.O. Box 5083, Boksburg North.
123. South Atlantic Corporation Medical Aid Society, P.O. Box 4610, Cape Town.
124. Southern Medical Aid Society, P.O. Box 42, Cape Town.
125. Standard Brass Medical Aid Society, P.O. Box 229, Benoni.
126. Steeldale and Union Joinery Medical Aid Society, P.O. Box 1210, Johannesburg.
127. Stewarts & Lloyds Medical Benefit Fund, P.O. Box 74, Vereeniging.
128. Stuttafords Medical Aid Society, P.O. Box 69, Cape Town.
129. Sun Insurance Office Ltd. Staff Medical Aid Fund, P.O. Box 429, Johannesburg.
130. Syfret's Medical Aid Society, 24 Wale Street, Cape Town.
131. Traduna Medical Aid Fund, P.O. Box 8791, Johannesburg.
132. Transvaal Corundum Associated Asbestos Medical Aid Society, P.O. Box 72, Pietersburg, Transvaal.
133. Transvaal Society of Accountants Medical Aid Fund, P.O. Box 2995, Johannesburg.
134. U.L.A. Medical Aid Society, P.O. Box 4589, Johannesburg.
135. Umzimkulu Sugar Co. Ltd. Medical Aid Fund, P.O. Box 43, Durban.
136. United Banks' Medical Aid Society, P.O. Box 1242, Cape Town.
137. United Building Society Medical Aid Fund, P.O. Box 7735, Johannesburg.
138. University of the Witwatersrand (Johannesburg) Staff Medical Aid Fund, Milner Park, Johannesburg.
139. Vacuum Medical Aid Society, P.O. Box 35, Cape Town.
140. Village Board of Management of Welkom Medical Aid Society, P.O. Box 708, Welkom, O.F.S.
141. Wright, Boag & Head, Wrightson Sick Benefit Fund, P.O. Box 183, Benoni.
142. Yorkshire Medical Aid Society, P.O. Box 2755, Johannesburg.

MEDICAL BENEFIT SOCIETIES WHICH ALLOW FREE CHOICE OF DOCTOR FOR SPECIALIST SERVICES ONLY
MEDIESE BYSTANDSVERENIGINGS WAT VRY KEUSE VAN DOKTER ALLEEN VIR SPESIALISTEDIENSTE TOELAAT

1. Begbie Medical Benefit Fund, P.O. Box 192, Middelburg, Transvaal.
2. Brakpan Power Station Sick Benefit Society, P.O. Box 1, Brakpan.
3. Breyten Coalfields Benefit Society, P.O. Box 6, Estantia, Transvaal.
4. Broken Hill Mine Employees' Medical Specialist Fund, P.O. Box 45, Broken Hill.
5. De Beers Consolidated Mines Limited Benefit Society, P.O. Box 616, Kimberley.
6. Durban Roodepoort Deep Ltd. Benefit Society, P.O. Box 193, Roodepoort.
7. Jagersfontein Mine Benefit Society, P.O. Box 2, Jagersfontein, O.F.S.
8. Krugersdorp Municipal Employees' Medical Benefit Society, P.O. Box 101, Krugersdorp.
9. Northern Rhodesia Mine Employees Medical Specialist Fund, P.O. Box 134, Kitwe, Northern Rhodesia.
10. Public Utility Transport Corporation Sick Fund, P.O. Box 9571, Johannesburg.
11. Randfontein Estates Employees' Sick Benefit Society, P.O. Box 37, Randfontein.
12. Roodepoort-Maraisburg Municipal Employees' Association Sick Benefit Society, P.O. Box 217, Roodepoort.
13. Roodepoort-Maraisburg Non-Scheduled Mines' and Industries' Benefit Society, P.O. Box 225, Roodepoort.
14. Rosherville-Maraisburg Benefit Society, P.O. Box 99, Cleveland, Johannesburg.
15. Sasol Medical Benefit Society, P.O. Box 80, Sasolburg.
16. Simmer Pan Medical Benefit Society, P.O. Box 103, Germiston.
17. Springs Mines Benefit Society, P.O. Box 54, Springs.
18. Tongaat Sugar Company Medical Benefit Scheme, P.O. Box 5, Maidstone, Natal.
19. Transvaal Jewellers' & Goldsmiths' Sick Benefit Fund, P.O. Box 8530, Johannesburg.
20. Tweefontein Colliery Employees' Benefit Society, Tweefontein Colliery, P.O. Coalville, Transvaal.
21. Witbank Coalfields Benefit Society, P.O. Box 26, Witbank.
22. Witbank Power Station Medical Benefit Society, P.O. Box 197, Witbank.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

HUMEVAC CAPSULES

Parke, Davis Laboratories (Pty.) Ltd., in introducing a new type of non-cathartic faecal softener, Humevac capsules, supply the following information: Each Humevac capsule contains 250 mg. of the polymer of propylene oxide and ethylene oxide, which acts as a surface-tension depressant permitting fluids in the colon to penetrate into, and soften, hard dry faecal matter. This results in a softer and more homogeneous stool which is easily evacuated by normal peristalsis. Humevac is not in itself a laxative, bulk-producer or lubricant, nor does it possess any of the disadvantages associated with laxatives. The beneficial effect of Humevac is based on its ability to soften the stool by a wetting action, which is gradually accomplished after treatment is initiated.

Indications. Humevac is not intended to be drastic in action and thus has a wide field of application in many conditions requiring maintenance of soft stools, such as, for example, before and after surgery, during pregnancy and convalescence, for invalids and bedfast patients. Humevac is also useful as an aid in preventing or relieving constipation caused by iron therapy or barium retention enemas used during radiography. It is also indicated in the symptomatic relief of diverticulosis, where the reduction of surface tension may aid in helping food to slide out of the diverticulum, reducing the tendency for gas formation.

Dosage and administration. Orally: *Adults*—one capsule three times a day until the condition is corrected; one capsule daily thereafter as required. *Children*—one capsule daily until the

condition is corrected. Each dose should be followed by a glass of water. *Infants*—contents of one capsule mixed with milk, fruit juice, or solid food.

Package information. Humevac capsules, 250 mg., are supplied in bottles of 30 and 100.

TRILAFON REPETABS

Scherag (Pty.) Ltd. supply the following information:

Schering Corporation announces a new form of their well-known tranquillizer and anti-emetic, Trilafon. In keeping with the modern trend to avoid repeating doses, Trilafon is now compound as a Repetab, each tablet containing 8 mg. of perphenazine. The dosage is equally divided—4 mg. in an outer layer for immediate absorption, and 4 mg. in an inner core for release approximately 4-6 hours after ingestion.

Advantages. Trilafon Repetabs extend the tranquillizing and anti-emetic effects of Trilafon throughout the day or night following a single dose. They are of particular value in conditions or situations when an uninterrupted therapeutic effect from a single tablet is desired and when maintenance dosage has been established. Trilafon Repetabs assure continuous medication by minimizing the risk of 'forgotten' doses and relieve distressed patients of dosage worries and complicated directions. In hospitals they relieve nursing personnel of extra work.

Indications

Trilafon Repetabs are indicated especially when a prolonged tranquillizing effect is desired in the management of anxiety, tension and psychomotor overactivity. They are also highly effective in providing prolonged anti-emetic effect in the control of hyperemesis gravidarum and simple nausea and vomiting of pregnancy and of nausea and vomiting due to gastro-enteritis, post-operative states, carcinomatosis, drug or radiation therapy, and psychogenic factors.

Dosage

As with all potent drugs, the best dose is the lowest dose which provides the desired clinical effect, and each patient should be treated individually.

Simple anxiety and tension states. One Trilafon Repetab administered once or twice daily is generally adequate. A total daily dose of more than 2 Trilafon Repetabs is seldom required to elicit a favourable response.

Moderately disturbed out-patients. One or two Repetabs morning and night provides the usual total daily dose of 8-32 mg. in ambulant psychiatric out-patients.

Hospitalized Psychiatric Patients. Two to four Repetabs morning and night, according to severity of symptoms and individual response, is the usual dosage range. Total daily dose of 64 mg. should not be exceeded.

Nausea and Vomiting. Two Repetabs may be administered initially in acute cases. The average dose is one Repetab administered morning and evening.

In Children. One Trilafon Repetab may be given morning and evening to children over 12 years of age to provide tranquillization or anti-emetic effect.

Precautions and Contra-indications

Patients receiving Trilafon should be kept under regular observation. Agranuloctosis has not been reported with Trilafon, but the possibility of bone-marrow depression such as has been observed with other phenothiazine drugs cannot be ruled out. Leukopenia or other evidences of bone-marrow depression are contra-indications to the use of the drug. The anti-emetic effect of Trilafon may obscure signs of toxicity due to overdosage of other drugs or render more difficult the diagnosis of disorders such as brain tumour or intestinal obstruction.

Trilafon is contra-indicated in depressed conditions, whether psychic in origin or resulting from depressants of the central nervous system such as barbiturates, alcohol, narcotics, or similar drugs.

A significant rise in body temperature not otherwise explained may suggest individual intolerance, in which event the drug should be discontinued.

As with other phenothiazine drugs, Trilafon should be used with great caution in patients with a history of convulsive disorders, and patients who have exhibited severe side-actions to other phenothiazine drugs should be under constant supervision.

Extrapyramidal symptoms have been observed at times closely simulating the Parkinson syndrome, but these manifestations have disappeared within 48 hours on decrease in dosage or withdrawal of the drug or, if necessary, the administration of anti-Parkinson drugs.

Further information and trial material may be obtained from Scherag (Pty.) Ltd., P.O. Box 7539, Johannesburg.

REVIEWS OF BOOKS : BOEKRESENSIES

AIDS TO ORGANIC CHEMISTRY

Aids to Organic Chemistry for Medical Students. 5th Edition. By George A. Maw, Ph.D., F.R.I.C. Pp. vii+176. 10s. 6d. London: Baillière, Tindall and Cox Ltd. 1958.

The last example in the 'Aids' series—that support of the student in the days of his training—to appear is the 'Aid to Organic Chemistry'. As ever, the information is carefully selected and packed. The coverage is thorough and the knowledge concentrated. The medical student will find that he will not be let down by this representative of the series.

T.S.

REHABILITATION AFTER ILLNESS

Rehabilitation after Illness and Accident. Edited by Thomas M. Ling, M.D., M.R.C.P. and C. J. S. O'Malley, C.B.E., M.B. Pp. vii+119. 12s. 6d. London: Baillière, Tindall and Cox Ltd. 1958.

All the contributors to this excellent work are or have been in some way associated with St. Thomas's Hospital, London. It is a product of which this seat of teaching and healing may well be proud and it is greatly to be regretted that Group Captain O'Malley, the co-editor, did not live to see the book from the printing press. The rehabilitation and resettlement of the disabled is a subject which is evoking increasing interest not only among medical practitioners and workers in ancillary fields, but its prime importance is being recognized equally by social workers and industrialists.

In this book authoritative opinion in all the important branches of the subject is collected and expressed with wisdom and practical good sense. The difficulties to be overcome by the mentally and physically disabled are dealt with in detail and there are few aspects of the problem that do not receive well-balanced and imaginative consideration. Stress is laid upon the essential need for teamwork and education in the task of helping handicapped persons in their struggle towards social and industrial independence.

Physical procedures in the field of rehabilitation are well known and generally accepted. One is struck, however, by the emphasis placed by all the contributors on the psychological and emotional factors influencing recovery of function and the restoration to a useful and productive life of the sick and injured. In my opinion there are few works more capable of elevating the level of practice in all branches of medicine. The book is strongly recommended.

M.G.W.

AUTONOMIC IMBALANCE

Autonomic Imbalance and the Hypothalamus. Implications for Physiology, Medicine, Psychology and Neuropsychiatry. By Ernst Gellhorn, M.D., Ph.D. Pp. xiv+300. 101 Figures. English Price, approximately 48s. Minneapolis: University of Minnesota Press. 1958. Local Distributors: Oxford University Press.

This book contains two parts. Part I, the experimental section, deals with autonomic 'tuning', and describes states of autonomic imbalance produced by altering the excitability of the hypothalamus, through physiological and pharmacological means. In Part II the author discusses the clinical significance of sympathetic and of parasympathetic predominance, in health and disease.

The book is well written and stimulating, because it challenges the adequacy of both the older, and well-known, theories and classifications of psycho-physiological reactions and functional disorders. It should be of interest to both students and teachers of psychology, psychiatry and medicine.

A.B.v.d.M.

THE THYROID AND PARATHYROID GLANDS

Diseases of the Thyroid and Parathyroid Glands. By Bernard J. Ficarra, A.B., Sc.B., M.D., D.S. Pp. viii+295. 131 Figures. $8·50. New York: Intercontinental Medical Book Corporation. 1958.

The title of this book is misleading. Anyone who expects a full and comprehensive dissertation on all the diseases of these two endocrine glands is bound to be disappointed. For it is a book which aims only at presenting the accumulation of knowledge gained by an average general surgeon with an average general practice in an average American community. And yet it wanders sufficiently far into realms of internal medicine to make one feel that a little more attention to the 'medical' aspects would have vastly improved this book. One misses for instance an adequate account of myxoedema and a dissertation on hypoparathyroidism (apart from the post-operative variety). The medical management of thyrotoxicosis is not adequately dealt with—the author all too obviously prefers surgery—and the section on the management of exophthalmos could also be vastly improved by a less radical approach. Nevertheless, if one approaches the book from the point of view of a surgeon, it has much of interest.

The author has obviously had a vast surgical experience of thyroid disease and knows all the practical aspects which he needs to know. The section dealing with operative and post-operative management are good and so are those dealing with the complications of operation. Thyroid crisis is extensively considered as are thyroid cancer, ectopic thyroids and other thyroid anomalies. And if he does move from one subject to another in a rather irregular sort of way, perhaps he may be forgiven for he has much of his own experience to offer. There is even a little section entitled 'Chronologic résumé of the thyroid and parathyroid history' starting with the use of burnt seaweed and sponges in wine (by the ancient Chinese in 1600 B.C.) for the treatment of goiter.

The book is nicely produced and well illustrated. Embryo surgeons could learn much from the author. Other practitioners would also benefit from it if they don't start off on the wrong foot and expect something else.

M.C.

CORRESPONDENCE : BRIEWERUBRIEK

SKF LABORATORIES AWARD FOR POSTGRADUATE CLINICAL STUDY IN SOUTH AFRICA

To the Editor: This Award was established by SKF Laboratories (Pty.), Ltd., P.O. Box 784, Port Elizabeth, which is the South African branch of Smith, Kline and French Laboratories Ltd., London. The value of the Award is £200. It requires the successful candidate to spend a minimum period of 2 months doing postgraduate clinical work at any institution or medical school approved by the Selection Committee.

The Selection Committee consisted of the following: Prof. J. F. Brock (Cape Town), Prof. G. A. Elliott (Johannesburg), Dr. H. A. Shapiro (*Honorary Chairman*, Johannesburg), Dr. M. Shapiro (Johannesburg), Dr. M. M. Suzman (Johannesburg), and Prof. H. W. Snyman (Pretoria).

The first recipient of this Award is Dr. D. I. L. de Villiers, of Standerton. Dr. de Villiers will spend a 2-month period of postgraduate study at the University of Pretoria.

H. A. Shapiro

P.O. Box 1010, Johannesburg — *Honorary Chairman: Selection Committee*
17 October 1958

SUBUNGUAL HAEMATOMA

To the Editor: I wish to describe a method of releasing blood from a subungual haematoma, which I think is not generally known. I claim no originality for the method, having learned it from the correspondence columns of the *Lancet* some years ago.

A wire paper-clip is opened out and held in artery forceps, and the last ½ inch or so is heated red hot. This is immediately used to pierce the nail, the underlying pool of blood preventing one from burning the patient. The hot wire melts an adequate hole in the nail, from which the blood streams immediately.

The operation is painless, rapid and effective, and to my mind, far superior to any other method I have seen described.

Seymour Dubb

Rondebosch, Cape Town
18 October 1958

CLINICAL SPECIALITIES: PATHOLOGY IN LIEU OF GENERAL PRACTICE

To the Editor: During a recent visit to Canadian and United States laboratories I was impressed by the fact that a considerable proportion of clinicians wishing to specialize in Surgery, Medicine, Paediatrics or other clinical subjects spend a year in a Department of Pathology. The benefits of the arrangement were emphasized to me by the clinical registrars, who feel that it enables them to obtain an invaluable basic knowledge of the pathogenesis, morbid anatomy and histopathology of disease in their chosen field. On the other hand the pathologists feel that the year spent in pathology gives clinical registrars an insight into pathology and its problems which is otherwise unobtainable. The year in pathology is also of value to the clinician when he is planning future research programmes. It is my own opinion, and that of several pathologist colleagues and clinical registrars to whom I have spoken, that it is unfortunate that advantage is not taken of a similar system of training in South Africa.

Perusal of the South African Medical and Dental Council's 'Rules concerning registration of specialities shows that advantage could be taken of such a system in South Africa under existing regulations if the clinicians and pathologists wished to do so. Under Rule 5 (*c*) Note (2) it is permissible for an applicant for registration in a clinical speciality to offer one year's service in Pathology in lieu of general practice. This interpretation of Rule 5 (*c*) is confirmed by the Acting Registrar of the South African Medical and Dental Council who, at my request, placed the matter before the Council. The Acting Registrar replies as follows in a letter dated 22 July 1958:

'I am directed by my Committee to forward you a copy of the Council's rules for the registration of specialities, which I append hereto, and invite your attention to the rule relating to experience in lieu of general practice. In terms of this rule it is permissible for an applicant for registration of all specialities to offer one year's service in Pathology as service in lieu of general practice; what you suggest in your letter is consequently already permissible under the rules.'

The scheme holds such advantages for practitioners in clinical medicine and pathology alike that I crave the use of your columns to bring it to the notice of interns, general practitioners and registrars who are contemplating specialization in a clinical subject. Judging by the infrequency with which clinical registrars seek experience in Pathology it seems likely that they are unaware of the provision which Medical Council has wisely made for it as an optional part of their training.

J. F. Murray

S.A. Institute for Medical Research — *Medical Superintendent*
P.O. Box 1038, Johannesburg — *Routine Diagnostic Division*
18 October 1958

CAPE TOWN MEDICAL ART SOCIETY

To the Editor: On 29 September a small group of medical men met and formed themselves into the Cape Town Medical Arts Society. The aim of this Society is to promote interest and enjoyment in the arts and encourage creative work amongst medical and dental practitioners. The Society proposes to hold a public exhibition of its work annually. A Steering Committee was elected, consisting of Dr. J. McGregor, Dr. A. M. Whitaker and Dr. C. W. Coplans (Hon. Secretary). Medical men and dentists in Cape Town who would be interested in joining the Society should communicate with me.

C. W. Coplans

906 Sam Newman House
28 Burg Street, Cape Town
15 October 1958.